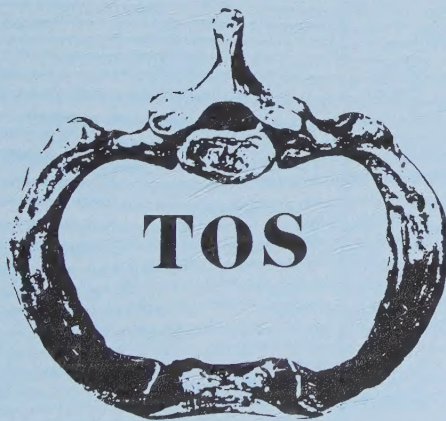


The thoracic outlet syndrome

*A Clinical Study with Special Reference to
Diagnosis, Incidence, and Treatment*

By

JOHAN SÄLLSTRÖM



hund 1983



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Depts of Surgery and Clinical Physiology Central Hospital, Växjö and dept of
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MALMÖ 1983



The English text has been revised by Stanley H. Pretorius

The figures have been drawn by Ulla and Johan Sällström

Smålandspostens Boktr. AB, Växjö 1983

This dissertation is based on the following publications, which will be referred to by Roman numerals in the text:

- I. J. Sällström & O. Thulesius, 1982:
Non-invasive investigation of vascular compression in patients with thoracic outlet syndrome. Clin. Phys. 2:117-125, 1982.
- II. J. Sällström & H. Schmidt, 1983: Cervicobrachial disorders in certain occupations with special reference to compression in the thoracic outlet. Accepted for publication 1983; Am. J. Ind. Med.
- III. J. Sällström & Z. Celegin, 1983: Physiotherapy in patients with thoracic outlet syndrome. Accepted for publication 1983; VASA
- IV. J. Sällström & J.E. Gjörres, 1983: Surgical treatment of the thoracic outlet syndrome. Accepted for publication 1983; Acta Chir. Scand.

In the text, the thoracic outlet syndrome will be abbreviated TOS.

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Chapter One

Introduction

The thoracic outlet syndrome (TOS) is a symptom complex of neurovascular origin. The brachial plexus and the subclavian vessels leave the thoracic cage through the narrow thoracic outlet to the arm. If the neurovascular structures become compressed in this region, symptoms will occur in the head, neck, shoulder, and arm regions. There are a number of other diseases giving similar symptoms in the same regions, however. When establishing the diagnosis of TOS, high demands are made on the physician's knowledge of other possible diseases affecting the above-mentioned regions. A careful history and examination of the patient must be performed and all differential diagnoses must be excluded before the diagnosis of TOS can be established. This procedure is time-consuming and also difficult. Therefore, many attempts have been made to facilitate the diagnosis by means of objective tests.

For many decades, compression of the artery has been regarded as the main cause of TOS (Adson & Coffey, 1927; Naffziger & Grant, 1938). The close proximity of the artery to the brachial plexus has created a general belief that compression of the plexus was directly correlated to arterial compression. Accordingly, the diagnosis of TOS was established if arterial compression could be demonstrated with the arm in provocative positions. Few studies have been performed on the possible correlation between vascular and brachial plexus compression.

Increasing evidence indicating that compression of the brachial plexus is the main cause of TOS has been gathered. This compression is responsible for symptoms in almost 98 % of all patients with TOS (Roos, 1976). This has resulted in a number of neurophysiological studies assessing the ulnar nerve conduction velocity and the sensory action potentials (Urschel & Razzuk, 1972; Sadler *et al.*, 1975; Wulff & Gilliat, 1979; Glover *et al.*, 1981).

Anatomical considerations

The thoracic outlet

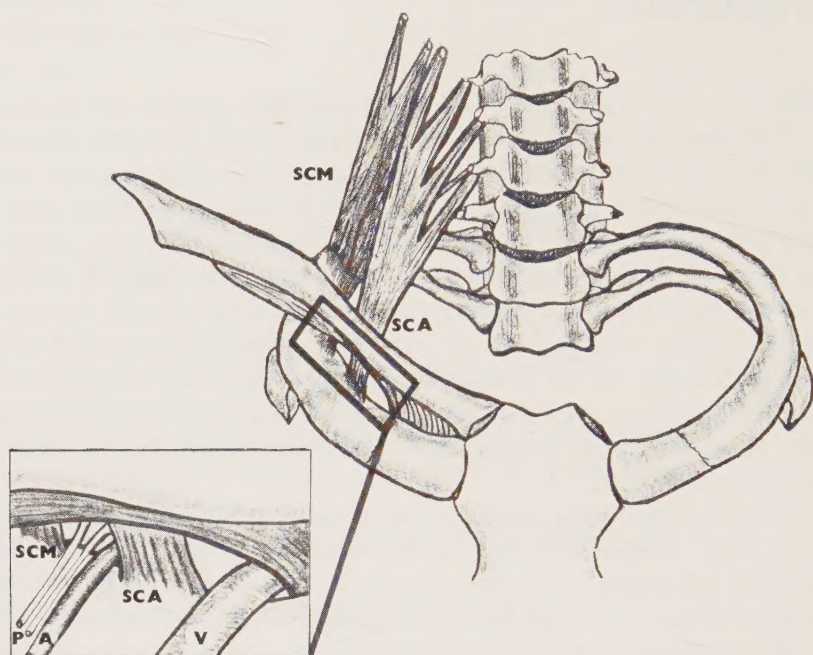


Fig. 1. Anatomy of the thoracic outlet. SCM = Scalenus medius muscle; SCA = Scalenus anterior muscle; P = plexus brachialis; A = subclavian artery and V = subclavian vein.

The floor of the outlet consists of the first thoracic rib and the deep cervical fascia of Sibson. The latter is attached to the transverse process of C:7 and passes to the dome of the pleura and first rib lying deep to the neurovascular bundle. Superiorly, the outlet is defined by the clavicle and the subclavius muscle, anteriorly by the anterior scalene muscle, and posteriorly by the middle scalene muscle, see Fig. 1. The brachial plexus and subclavian artery emerge from between the anterior and middle scalene muscles and pass over the first rib. The lower trunk of the plexus lies directly behind the subclavian artery and is the most susceptible component of the plexus to compression. The subclavian vein is separated from the artery and the brachial plexus by the anterior scalene muscle. The vein passes through the triangular slit made up by the first rib inferiorly, the clavicle and the subclavius muscle superiorly, and the tendon of the subclavius muscle medially. The anterior scalene muscle constitutes the lateral border.

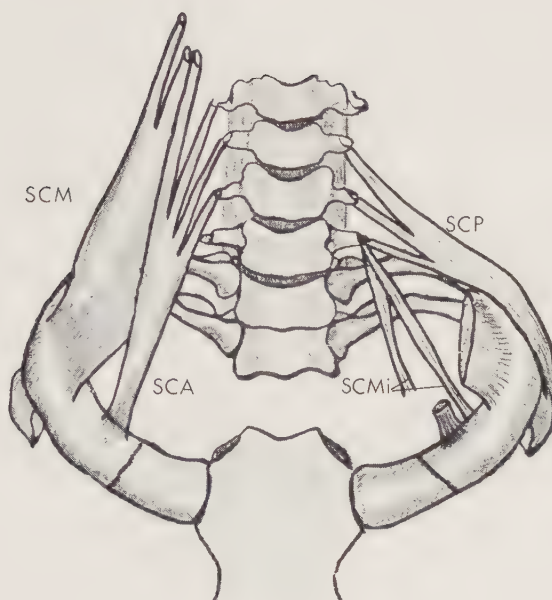


Fig. 2. The scalene muscles. SCM = Scalenus medius muscle; SCA = Scalenus anterior muscle; SCP = Scalenus posterior muscle; SCMi = Scalenus minimus muscle.

The scalene muscles

The anterior scalene muscle: This muscle arises from the anterior parts of the transverse processes of the 3rd, 4th, 5th, and 6th cervical vertebrae. The tendons join to form the belly of the muscle which inserts at the scalene tubercle of the first rib, see Fig. 2. Each tendon passes in close proximity to the cervical nerve root, emerging one segment below.

The middle scalene muscle: This muscle arises from the posterior parts of the transverse processes, starting at one vertebra above the anterior scalene muscle. The tendons fuse to the large belly of the muscle which attaches along the first rib with a broad insertion 1 cm behind the scalene tubercle, see Fig. 2.

The posterior scalene muscle: Arises from the posterior tubercles of the 5th, 6th, and 7th cervical vertebrae, passes downward behind the middle scalene muscle, and inserts at the second rib, see Fig. 2.

The minimal scalene muscle: This is an inconstant small muscle arising from the transverse process of the 7th cervical vertebra. The muscle inserts either at the dome of the pleura, see Fig. 2, or sometimes at the first rib between the anterior and middle scalene muscles, see Fig. 2.

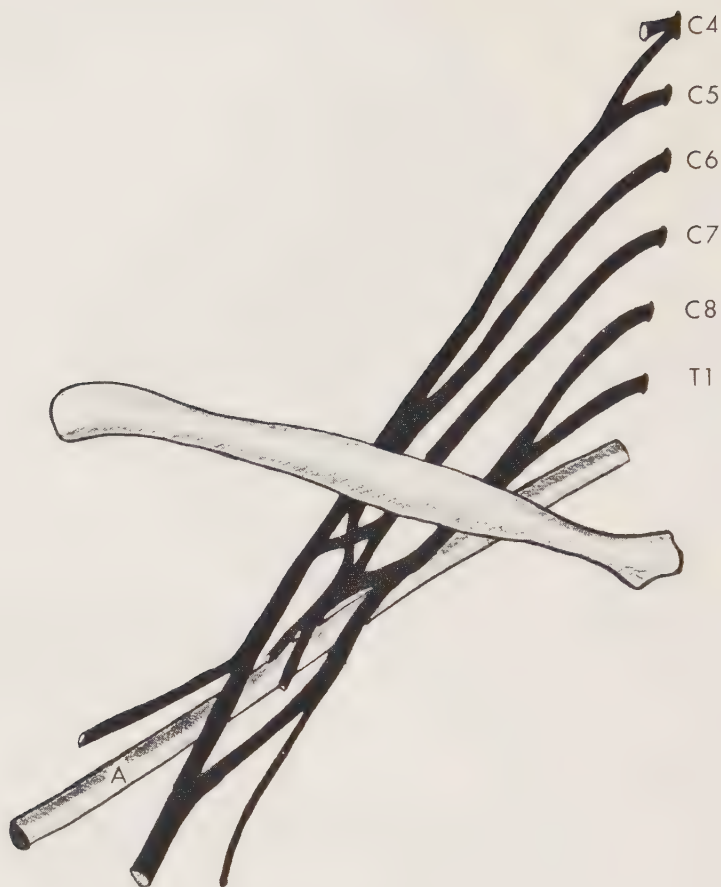


Fig. 3. The brachial plexus. A = subclavian artery.

The brachial plexus

The brachial plexus is formed by the union of the anterior primary rami of the lower four cervical and the first thoracic nerves, see Fig. 3. Close to the first rib, the roots of C:5 and C:6 combine to form the upper trunk, the C:7 root becomes the middle trunk, and the roots of C:8 and Th:1 unite to form the lower trunk of the brachial plexus. Each trunk divides into an anterior and a posterior division. Behind the clavicle, the nerves are rearranged into the lateral, medial, and posterior cords. The lateral cord is made up of the anterior division of the upper and the lower trunk, and all 3 posterior divisions unite to form the posterior cord. The cords are named after their location in relation to the axillary artery.

The plexus, together with the artery and the vein, pass beneath the pectoralis minor muscle before emerging into the superficial portion of the axilla. The final rearrangement of the cords to form the distal nerves takes place in relation to their passage beneath the pectoralis minor muscle and just distally to this area.

Branches from the medial and lateral cords form the *median nerve*. The remainder of the lateral cord becomes the *musculocutaneous nerve*, while the remainder of the medial cord becomes the *ulnar nerve*. The posterior cord splits into the *radial* and *axillary nerves*.

The fibers of C:5 are distributed mainly to the shoulder girdle muscles, to the flexors of the elbow, and the supinator of the hand. The fibers of C:6 re-inforce C:5. The C:7 fibers are distributed primarily to the extensor muscles, while the C:8 and T:1 fibers go to the medial aspect of the limb and to the flexors of the wrist and fingers.

Different components of TOS

Compression of the brachial plexus

For many years, interest has been focused on arterial compression as the cause of the symptoms. In the diagnosis of TOS, assessment of arterial compression, whether by non-invasive or invasive methods, has been fundamental. The concept of brachial plexus compression as the main cause of TOS, has slowly become more and more widespread, however, since Bramwell first suggested the association of brachial plexus compression with the first rib (Bramwell, 1903). Keen, Thorburn, and Morley have described patients in whom the lower trunk of the brachial plexus was compressed due to an abnormal first rib (Keen, 1907; Thorburn, 1907; Morley, 1913). Murphy was the first to point out that brachial plexus compression could be provoked by a normal first rib (Murphy, 1906). After excision of the rib, the neurological symptoms disappeared completely. Stopford & Telford (1919), and Wheeler (1920), reported similar cases with a normal first rib as causative mechanism. Neurological symptoms may be elicited by cervical ribs, which was reported by Todd (1912). In 1920, Law described fibrous bands extending from the tip of an enlarged transverse process of a C:7 vertebra or cervical rib, passing behind and below the neurovascular structures, and compressing these structures against the anterior scalene muscle (Law, 1920). Law was the first to perform scalenotomy for the so-called cervical rib syndrome.

The descent of the shoulder aggravates the mechanical compression of the neurovascular structures according to Todd, (1912, 1922). Todd assumed that the shoulder girdle descends after birth due to the weight of the upper extremities. The relative position of the shoulders is higher in children and the shoulders descend gradually from birth to puberty to the adult position. Adson and Coffey stressed the importance of the anterior scalene muscle in the cervical rib syndrome (Adson & Coffey, 1927). The brachial plexus may be compressed between the anterior and middle scalene muscles, and Ochsner, Gage, and DeBaKey pointed out that the anterior scalene muscle can be responsible for pressure on the neurovascular structures against the first rib even without a cervical rib being

present (Ochsner *et al.* 1935). These authors believed that spasm in the anterior scalene muscle elevated the first rib and compressed the nerves and vessels against the clavicle. The middle scalene muscle has been considered the causative compression mechanism by several authors (Telford & Mottershead, 1947; Kirgis & Reed, 1948; Rogers, 1949; Stammers, 1950).

Swank and Simeone pointed out that both the upper and the lower trunks of the brachial plexus could be compressed (Swank & Simeone, 1944). Contraction and spasm of the posterior part of the anterior scalene muscle may compress the upper roots of the plexus. Lower trunk compression was elicited through penetration of the contracted muscle by the nerves (Swank & Simeone, 1944).

The costoclavicular syndrome was described in 1943 by Falconer and Weddell who reported that the compression in the thoracic outlet was caused by narrowing of the space between the clavicle and the first rib (Falconer & Weddell, 1943). Neurovascular symptoms could also be provoked by hyperabduction of the arms (Wright, 1945). The neurovascular structures are stretched around the coracoid process and the pectoralis minor tendon at hyperabduction (Wright, 1945).

Rosati & Lord (1961) made an extensive review of the possible mechanisms of nervous compression. In addition to cervical ribs, anomalies of the first rib, and congenital bands, they described other mechanisms, e.g. the scalenus minimus muscle which arises from the transverse process of the C:7 vertebra and inserts at the first rib between the scalenus medius muscle and the scalenus anterior muscle, see Fig. 2. This muscle may compress the lower trunk of the brachial plexus against the scalenus medius muscle. Since then, increasing evidence has been gathered on the compression of the plexus as the main cause of TOS. Clagett (1962) redirected interest to the first rib as the main cause of compression in the thoracic outlet region. Several reports have been published on resection of the first rib causing relief of symptoms (Falconer & Li, 1962; Roos, 1966; Roos & Owens, 1966; Sanders *et al.*, 1968; Roos, 1971; Urschel & Razzuk, 1972; Dale & Lewis, 1975; Roos, 1976; Martinez, 1979; Thomas, 1979; Urschel, 1979; Hempel *et al.*, 1981). Roos reported congenital anomalies in the thoracic outlet and described nine different bands (Roos, 1976; Roos, 1979). These bands mechanically compress the neurovascular structures in the outlet, and Roos concluded that most patients with TOS have anomalous fibrous or muscular bands predisposing them to plexus compression.

Arterial compression

A diagnosis of *cervical rib syndrome* was first reported by Willshire (1860) and the first operation for this syndrome was performed by Coote in 1861 when he resected a cervical rib by the supraclavicular approach (Coote, 1861).

In 1905, Murphy performed the first cervical costectomy of a cervical rib in the presence of a subclavian aneurysm (Murphy, 1905). He was the first to advance the theory of neurovascular bundle compression between a cervical rib and the anterior scalene muscle.

The etiology of vascular symptoms has been a much debated subject. The vascular lesion may be caused by pressure on the subclavian artery, either by a cervical rib or by the anterior scalene muscle (Adson & Coffey, 1927; MacFee, 1940). Furthermore the compression can be elicited by nipping of the artery between the clavicle and the rib (Eden, 1939; Falconer & Weddell, 1943; Walshe *et al.*, 1944).

In 1919, Stopford and Telford advocated the theory that irritation of the sympathetic nerves as they run in the brachial plexus is the cause of the vascular symptoms (Stopford & Telford, 1919). The ischemic attacks were considered to be vasospastic phenomena, and the local damage to the artery was assumed to be caused by spasm of the vasa vasorum resulting in degeneration of the arterial wall (Stopford & Telford, 1919; Telford & Mottershead, 1947). In 1934, Lewis and Pickering pointed out that "all the vascular symptoms displayed could not arise directly out of compression or out of permanent obstruction of the subclavian artery, but they could arise out of thrombosis with embolism" (Lewis & Pickering, 1934).

Rosati and Lord (1961) proposed the theory that vascular symptoms are caused by a combination of direct arterial compression and vasomotor nerve irritation in varying degrees.

Judy and Heyman (1972) stressed that a history suggestive of peripheral embolization, i.e. unilateral Raynaud's phenomenon and fingertip ulceration episodes with ischemic pain, is a forerunner of a more severe problem. The concept of vascular compression as the main cause of the thoracic outlet syndrome has been increasingly debated (Dale & Lewis, 1975; Rainer & Sadler, 1975; Roos, 1976; Gergoudis & Barnes, 1980; Dale, 1982; Martinez, 1982b).

Venous compression in the thoracic outlet

In 1924, Lowenstein found at necropsy that during abduction, extension, and lateral rotation of the arm, the axillary vein comes into close contact with the lateral border of the costocoracoid membrane, i.e. the costocoracoid ligament and the subclavian muscle. He assumed that this condition, in connection with the venous stasis during vigorous muscle exertion, gives rise to changes in the vessel wall (Lowenstein, 1924). In patients with pronounced compression of the subclavian vein, McLaughlin and Popma considered that the vein was pressed against the clavicle by a taut anterior scalene muscle and recommended scalenotomy (McLaughlin & Popma, 1939; Sampson *et al.*, 1940). In 1940, Veal called attention to the constricting pressure effect of the head of the humerus through the subscapular muscle in the abduction position of the arm (Veal, 1940). In 1945, Wright described the hyperabduction syndrome. He believed that the vein was damaged in the abducted position of the arm as it passed around the coracoid process and the tendon of the pectoralis minor muscle (Wright, 1945).

McCleery *et al.*, reported that the subclavius muscle, the costocoracoid liga-

ment, or the anterior scalene muscle—or a combination of these—were the causative mechanism in venous compression (McCleery *et al.*, 1951).

Rosati and Lord studied patients suffering from “effort-thrombosis” (Rosati & Lord, 1961). They thought the obstruction point was at the site where the vein passes between the costocoracoid ligament and the subclavius muscle anteriorly and the first rib posteriorly. The obstruction was caused by a changed relationship between the shoulder girdle and the thoracic outlet caused by sagging of the shoulder girdle. In such cases, sustained effort with the arm in abduction, sudden backward and downward bracing of the shoulders, heavy lifting, or strenuous physical activity involving the arm, may suddenly constrict the vein and initiate venospasm with or without subsequent thrombosis (Rosati & Lord, 1961). In 1976, Ross pointed out that the symptoms of venous compression are quite rare, present only in about 1 1/2 % of all patients with TOS, although they are more frequent than symptoms of arterial insufficiency (Roos, 1976). Compression of the subclavian vein is not correlated to TOS as the vein is completely separated from the artery and brachial plexus by the anterior scalene muscle.

Pathophysiology and incidence

Pathophysiology

A number of acquired and congenital factors may elicit TOS:

Acquired factors

Trauma

A fractured clavicle with considerable callus formation or osteochondroma of the first rib or other factors causing an increase in the bony substances in the outlet area may cause TOS symptoms. Traumas to the head, neck or shoulders are quite common as TOS-eliciting factors which has been pointed out by several authors (Roos, 1971; Dale & Lewis, 1975; Roos, 1976; Capistrant, 1977; Kelly, 1979; Sanders *et al.*, 1979; Martinez, 1982a).

Whiplash injuries seem to be overrepresented in TOS. Woods obtained a history of whiplash injury in 64 %, Sanders in 52 %, and Roos and Owens in 34 % of TOS patients (Woods, 1965; Roos & Owens, 1966; Sanders *et al.*, 1968).

Postural and occupational factors

Poor posture with sagging shoulders is known to narrow the thoracic outlet region (Todd, 1922; Crawford, 1980). Pendulous breasts and muscular hypertrophy are also known causes. Certain occupations involving strenuous activities of the upper extremities or bad working positions provoke symptoms of the neck, shoulder, or arm regions (Nishiyama *et al.*, 1973; Maeda, 1975; Nakaseko *et al.*, 1975; Ohara *et al.*, 1976a; Ohara *et al.*, 1976b; Onishi *et al.*, 1976; Maeda, 1977). The incidence of TOS elicited by occupational factors is not known, however.

Congenital factors

Cervical ribs or anomalies of the first thoracic rib

A rudimentary first thoracic rib (Sargent, 1921) or a cervical rib are wellknown predisposing factors (Sargent, 1913; Davis & King, 1938; Hill, 1939; Etter, 1944; White *et al.*, 1945; Adson, 1951; Crimm, 1952; Schein *et al.*, 1956).

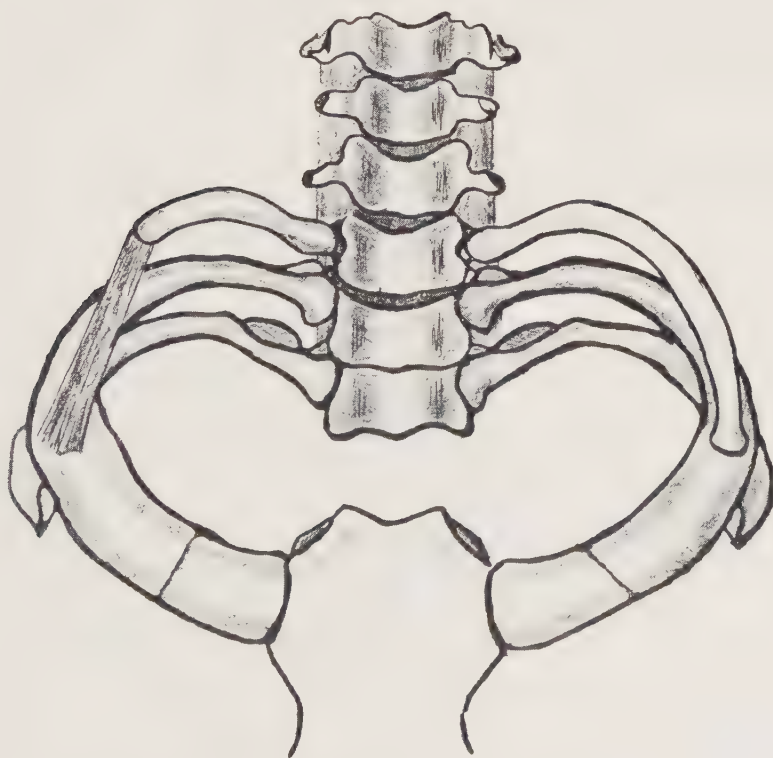


Fig. 4. Complete cervical rib with osseous union or incomplete rib with ligamentous attachment to the first rib.

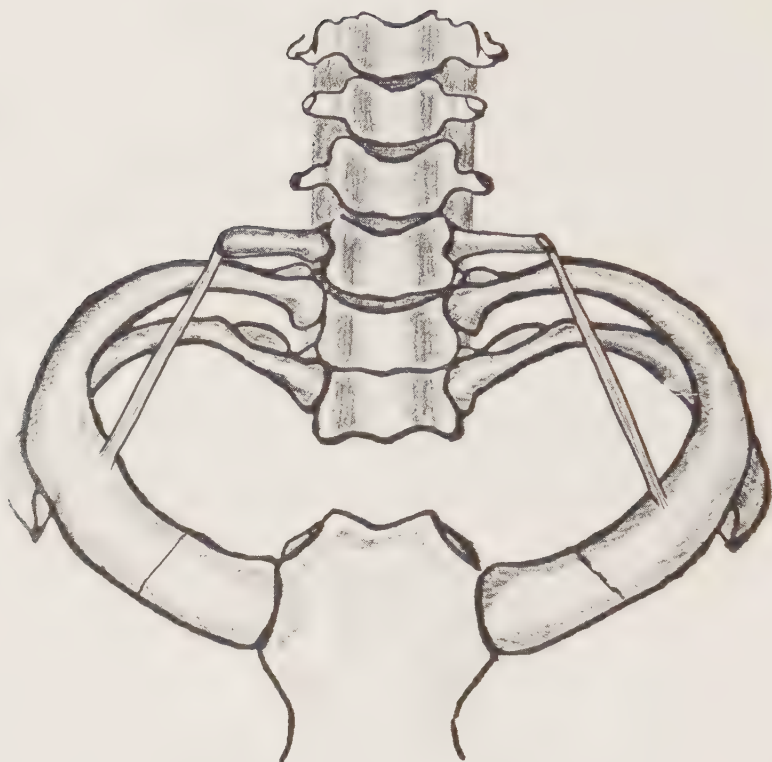


Fig. 5. Abortive cervical rib or prolonged transverse process with ligamentous attachment to the first rib.

Between 0.06 and 1 % of the population has either a prolonged transverse process of the 7th cervical vertebra or a cervical rib. The size of the rib varies from a small rib to a complete union between the cervical and first thoracic ribs at the region of the scalene tubercle, either by ligamentous or osseous attachment, see Fig. 4. Abortive cervical ribs or a prolonged C:7 transverse process may also elicit TOS symptoms as there is often a ligamentous extension attaching to the first rib, see Fig. 5. Eighty per cent of the cervical ribs are bilateral, but only about 10 % of all patients with cervical ribs develop compression symptoms of the neurovascular structures. The percentage of cervical ribs among patients operated for TOS varies from 1 % to 15 % (Sanders *et al.*, 1968; Johnson, 1974; Dale & Lewis, 1975; Roos, 1976).

Congenital bands

In 98 % of patients operated for TOS, Roos showed the presence of congenital fibrous or muscular bands that mechanically compressed the thoracic outlet (Roos, 1976; Roos, 1979). Nine different bands have been reported (Roos, 1976;

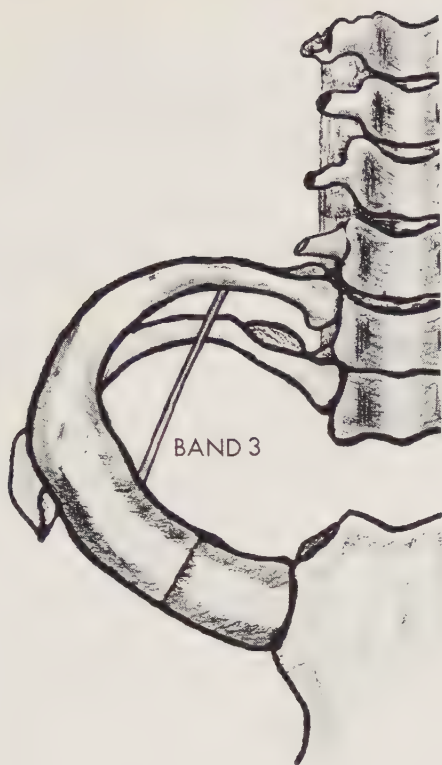


Fig. 6. Type 3 band, the most common congenital anomaly in the thoracic outlet.

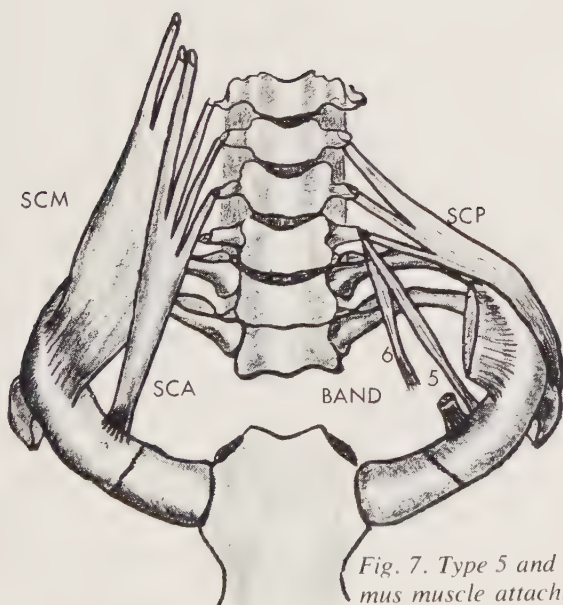


Fig. 7. Type 5 and 6 bands, i.e. the scalenus minimus muscle attaching either to the first rib or the cupola of pleura. SCM = scalenus medius muscle; SCA = scalenus anterior muscle; SCP = scalenus posterior muscle.

Roos, 1979). The most common anomaly is the Type 3 band, a fibromuscular structure traversing the inner curvature of the first rib in 60 % of patients operated for TOS, see Fig. 6. The next most common anomalies are the Type 5 and Type 6 bands which consist of the scalenus minimus muscle attaching either to the first rib between the middle and anterior scalene muscles or to the dome of the pleura, see Fig 7. In 17 % of patients operated for TOS, a Type 5 band is present. The Type 6 band is equally frequent. The other anomalies are present in 1–8 % of the patients (Roos, 1976).

The origins and attachments of the congenital anomalies as described by Roos 1979 are presented in Fig 8.

Type	Origin	Attachment
1	Ligamentous extension from incomplete cervical rib	First rib
2	Ligamentous extension from elongated C7 transverse pr	First rib
3	Fibromuscular band from neck of first rib	First rib behind scalene tubercle
4	Fibrous band from middle scalene muscle	Anterior scalene muscle under plexus and artery
5	Minimal scalene muscle	First rib between anterior and middle scalene mm.
6	Minimal scalene muscle	Cupola of pleura
7	Fibrous cord from anterior scalene muscle	Costochondral joint of first rib under the vein
8	Fibrous cord from middle scalene muscle	Costochondral joint of first rib under the plexus, artery, vein
9	Taut web of muscle and fascia inside first rib	Inside posterior curve of first rib, anterior sharp edge

Fig. 8. The congenital anomalies in the thoracic outlet; origins and attachments.

Incidence

A female predominance has been noted by several authors, the ratio being 2–3:1 (Thorburn, 1907; Adson & Coffey, 1927; Spurling & Bradford, 1938; Nelson, 1957; Clagett, 1962; Roos, 1966; Silver, 1968). Most patients are between 20 and 40 years of age.

The incidence of TOS is not exactly known, nor is it known if the incidence is higher in certain occupations or if certain activities or positions of the arms will increase the TOS incidence.

Diagnosis of TOS

Clinical symptoms

The symptoms may be entirely neurological, vascular, or a combination of both. The neurological symptoms are by far the most common ones which has been shown by a number of authors (Urschel & Razzuk, 1972; Dale & Lewis, 1975; Roos, 1976; Roos, 1977a; Roos, 1978; Youmans & Smiley, 1980; Martinez, 1982b).

Neurological symptoms: The most common complaint is pain in the supra-clavicular fossa, often radiating in all directions up the neck, back to the shoulder, forward to the chest, and down the arm and hand. The pain may be intermittent in the slight and moderate cases, or constant when the compression is more pronounced. The pain is often of an aching character with a sharp stabbing pain on provocation. Sometimes, the pain localized to the chest is misinterpreted as angina pectoris or even myocardial infarction (Urschel *et al.*, 1973). The second most common symptoms are paraesthesias and numbness. These symptoms most often involve the 4th and 5th fingers and the ulnar side of the forearm, and may be brought on by daily activities, but may also appear at rest during the night, awakening the patient. Furthermore, the patient often complains that the arm feels heavy and weak.

When the compression of the brachial plexus is pronounced, additional symptoms of progressive dysfunction of the hand will appear.

Circulatory symptoms

Arterial symptoms: These symptoms have been investigated most often mainly because arterial compression can easily be assessed objectively, either by non-invasive or invasive methods. In the past, much importance has been attached to arterial compression as being pathognomonical for TOS. A number of studies have shown that this is not the case, however (Wright, 1945; Raaf, 1955; Gilroy & Meyer, 1963; Dunant *et al.*, 1974; Roos, 1976).

In the very few patients with arterial compression leading to either arterial occlusion or aneurysmal formation, ischemic pallor of the hand at elevation is the most common symptom. In addition, coolness and ischemic pain in the hand as well as early muscle fatigue are quite common in these patients. There may also be finger tip ulcerations and necrosis due to peripheral embolization. This is sometimes misinterpreted as Raynaud's phenomenon. Such a misinterpretation may also appear in patients with a pain-induced increase in the sympathetic tonus similar to Raynaud's phenomenon.

Venous symptoms: Significant compression of the subclavian vein results in swelling and congestion. With the arm in provocative positions, the arm becomes

cyanotic with a blueish-red discoloration. If the compression is pronounced, distention and engorgement of the superficial veins appear across the shoulder (Adams *et al.*, 1968).

Common to all compression symptoms in the thoracic outlet is that they are intermittent and brought on by certain positions. Typically, the symptoms are provoked by using the arm, especially in elevation above the head. The symptoms are aggravated by the use and exercise of the arm, but very often the aggravation occurs after, rather than during, the exercise.

Clinical investigation

A careful history covering symptoms of weakness, pain, and paraesthesias at provocation in the affected limb is taken. Different tests, i.e. Adson, Eden, and Wright maneuvers have been used in the diagnosis of TOS.

In the Adson test, the patient takes a deep breath, elevates his chin and turns his head towards the affected side (Adson & Coffey, 1927; Adson, 1947; Adson, 1951).

In the Eden test, the patient braces his shoulder backwards (Falconer & Weddell, 1943) and in the Wright test, the patient puts his hands behind his head (Wright 1945).

These tests, however, although extensively used for years, add very little to the diagnosis of TOS (Wright, 1945; Telford & Mottershead, 1948; Eklöf, 1976; Roos, 1976; Longo *et al.*, 1979; Youmans & Smiley, 1980).

The Adson test has no relation to TOS (Roos, 1976; Youmans & Smiley, 1980). The Wright maneuver provokes arterial compression in 83 % (Wright, 1945) and the Eden test in 60 % of normal subjects (Falconer & Weddell, 1943; Raaf, 1955; Falconer & Li, 1962).

At neurological examination, sensory impairment in the ulnar aspect of the hand and forearm as well as weakness and fatigue of the arm are noted. Neurologic symptoms are provoked by positional changes and reproduction of symptoms is elicited by the elevated arm stress test advocated by Roos, see Fig. 9 (Roos, 1976). The reliability of this test has been questioned by Lord, who considered the test simply interfering with arterial flow to the upper extremity and not with the brachial plexus (Lord, 1981). Arterial compression can be provoked in normal subjects, however, *without* any TOS symptoms (Falconer & Weddell, 1943; Wright, 1945; Raaf, 1955; Falconer & Li, 1962; Gilroy and Meyer, 1963; Torriani, 1971; Dale & Lewis, 1975; Roos, 1976; Gergoudis & Barnes, 1980). Furthermore, symptoms can be provoked in the elevated arm stress test in patients with TOS regardless of arterial compression (Roos, 1976). Often, the brachial plexus is tender in the supraclavicular fossa. The vascular compression is assessed by digital palpation of the radial pulse and infraclavicular auscultation with the arm in different positions to detect bruits as signs of vascular compres-

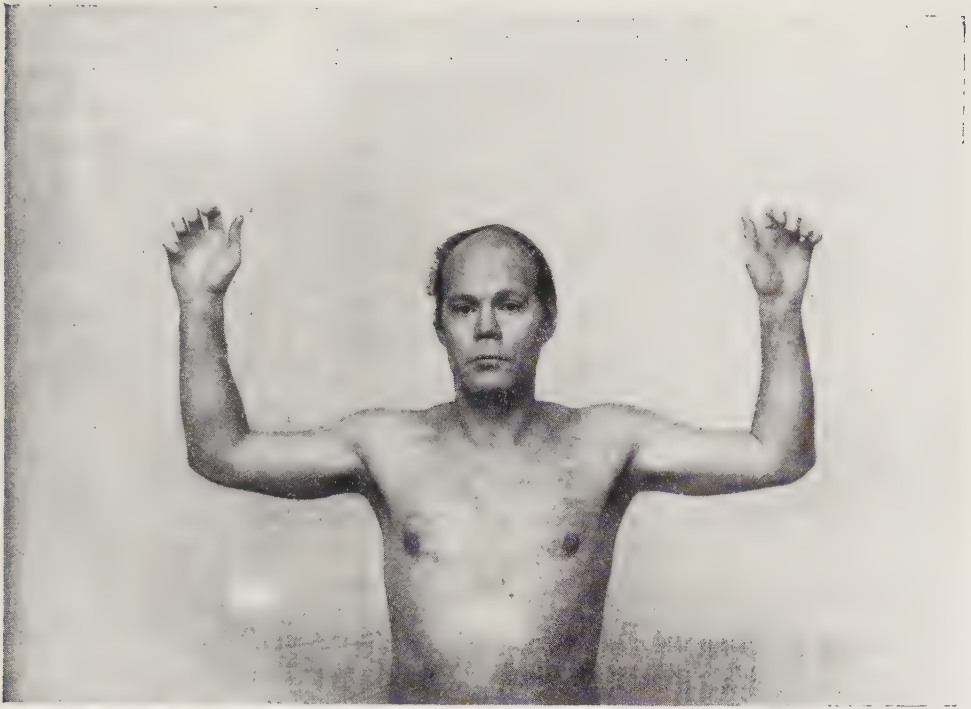


Fig. 9. The abduction-external-rotation (AER) position.

sion. This is the easiest way to evaluate arterial compression, but positional compression of the artery is a common phenomenon even in normal subjects (Wright, 1945; Raaf, 1955; Gilroy & Meyer, 1963; Torriani, 1971; Dale & Lewis, 1975; Roos, 1976; Gergoudis & Barnes, 1980).

Laboratory studies

Because the diagnosing of TOS is difficult and calls for a detailed history and physical examination, several attempts have been made to find an objective method of diagnosis. As the brachial artery lies close to the plexus, arterial compression has been thought to be directly correlated to compression of the brachial plexus. Because arterial compression is easy to detect and register objectively it has been used widely in the diagnosis of TOS.

Non-invasive assessment of arterial compression

Photoplethysmography, *oscillography* and *phonoangiography*, *plethysmography*, or *Doppler flowmetry* have been used to assess the vascular compression. Common to all these different tests, however, is the inability to predict concomitant plexus compression (Dunant *et al.*, 1974; Lo-A-Njoe, 1974; Hehne *et al.*, 1975; Dunant, 1976; Gergoudis & Barnes, 1980).

Invasive vascular tests

Positional selective subclavian arteriography has been advocated by several authors and has been used as the main diagnostic means in patients with TOS (Lang, 1965; Lang, 1966; Rosenberg, 1966; Nelson & Jenson, 1967; Weibel & Fields, 1967; Dick, 1970; Adler & Hooshmand, 1973). The incidence of arterial compression of clinical significance is extremely low. Furthermore, vascular compression can be provoked in the majority of normal subjects. Consequently, arteriography should not be used in the diagnosis of the thoracic outlet syndrome except in the very few patients with clinical signs of pronounced compression (Rainer & Sadler, 1975; Roos, 1976). Arteriography is indicated if

- a) the blood pressure difference is more than 15 mm Mercury between the symptomatic limb and the other side;
- b) there are prominent supraclavicular pulsations or
- c) supra- and infraclavicular bruits with the hand in the lap;
- d) radial pulse is absent or weak in the symptomatic limb with the hand in the lap;
- e) there is a suspicion of embolization to the fingers or hand;
- f) a bony anomaly is present, i.e. cervical rib or prolonged transverse process on the 7th cervical vertebra concomitant with bruits or arterial occlusion at provocation.

Neurophysiological tests

Nerve conduction studies have been used in the diagnosis of TOS. They have been found helpful by some (Urschel & Razzuk, 1972; Sadler *et al.*, 1975; Gilliat, 1976; Gilliat *et al.*, 1978; Stanton *et al.*, 1978; Urschel, 1979; Wulff & Gilliat, 1979; Glover *et al.*, 1981) but have been of little benefit to most others (Dale & Lewis, 1975; Daube, 1975; Kremer & Ahlqvist, 1975; Rainer & Sadler, 1975; Roos, 1976). However, these studies are very useful in the differential diagnosis of peripheral nerve compression, such as compression of the median nerve in the carpal tunnel and compression of the ulnar nerve in Guyon's canal and at the elbow joint (Jebsen, 1967; Caldwell *et al.*, 1971). Recently, there has been some interest in measuring the sensory action potentials (SAP) of the ulnar nerve (Gilliat *et al.*, 1978). The ulnar SAP is important in differing between peripheral entrapments and intraspinal lesions causing atrophy of innervated muscles in the hand. Ulnar SAP is normal in patients without atrophy in spite of the presence of cervical ribs, however (Gilliat *et al.*, 1978). In 1981, Glassenberg reported a new nerve conduction procedure where the ulnar nerve conduction was measured over

Erb's point in the elevated arm stress test (Roos' test) (Glassenberg, 1981). Erb's point is located in the supraclavicular fossa just laterally of the border of the sternocleidomastoid muscle and 2–3 cm above the clavicle at the level of the transverse process of the sixth cervical vertebra. By placing the electrode at Erb's point, the brachial plexus is stimulated. Abnormal nerve conduction was not considered necessary to justify the diagnosis and surgical treatment of TOS, however (Glassenberg, 1981).

In a few cases of TOS, Glover registered positive evoked potentials but negative in some cases (Glover *et al.*, 1981). Wulff & Gilliat found an increase in F wave latency in patients with atrophy of intrinsic hand muscles caused by a cervical rib. In patients with disabling pain and paraesthesias in the arms without hand wasting, however, the F wave characteristics were normal (Wulff & Gilliat, 1979).

So far, reliability of these tests to select patients with TOS is not sufficiently good to render them useful in the diagnosis of TOS. Most often, the compression of the brachial plexus differs from other nerve lesions, such as the carpal tunnel syndrome or the ulnar nerve entrapment at the elbow region, in that the compression is intermittent. This is not likely to give a reduction of the conduction velocity. Furthermore, the site of the compression makes it very difficult to place the stimulating electrode proximally to the lesion. The normal range is wide in that one laboratory interprets a specific conduction velocity as being indicative of TOS, while other laboratories interpret the same velocity as being quite in the normal range (Roos, 1976).

In conclusion—, no objective test has been developed that makes it possible to confirm the diagnosis of brachial plexus compression.

Differential diagnosis

Symptoms in the neck, shoulder, or arm regions are common. The most frequent symptoms are pain, paraesthesias, numbness, and weakness. There are numerous pain—sensitive structures in the cervical spine, the thoracic outlet, and the upper extremities, such as muscles, ligaments, joints, joint capsules, nerves, and vertebral bodies. Because of the multitude of structures which may be involved in the production of pain, patients complaining of pain in the upper extremities, shoulder, or neck regions present difficult diagnostic problems. Another problem is that the site where the patient experiences the pain may merely be the area to which the pain is referred. To reach and verify a diagnosis, a very careful history is mandatory as to the type, localization, character, incidence, and duration of all symptoms, as well as a thorough examination. Some important differences in the distribution of pain can help in the diagnosis.

Pain arising from the cervical spine is nearly always felt in the spine, though it may be projected to the shoulder and arm. It is evoked and enhanced by certain

movements of the neck, and is accompanied by tenderness and limitation of motions of the neck.

Pain of brachial plexus origin is experienced in and around the shoulder. It is induced by the performance of certain tasks with the arm and by certain positions. Most often, there is also a tenderness in the supraclavicular fossa. When the lower trunk of the brachial plexus is compressed, the pain radiates down the ulnar aspect of the arm to the hand and fingers.

Shoulder pain is localized to the shoulder joint and sometimes radiates down the outer side of the arm to the elbow, influenced by motions, localized tenderness in the joint region, and limitation of motion.

Unilateral pain in the arm, shoulder, or neck always has an organic cause. Sensory or reflex changes indicate nerve involvement.

The cause of symptoms in the neck, shoulder, and arm-regions will be discussed under the following headings:

- I. Nervous origin
- II. Myogenic origin
- III. Miscellaneous origins

I. Nervous origin

Intraspinal cervical lesions

Description: Various extramedullary and intramedullary processes damaging nerve roots and/or the spinal cord, i.e. extramedullary cervical disc herniation, meningiomas, neurinomas, or intramedullary gliomas. Processes of the bony spinal column, such as primary sarcomas, multiple myeloma, or secondary metastases are also included in this group.

Symptoms:

- a) Pain, paraesthesias, paresis, and diminished sensibility correlated to the nerve root or segment involved.
- b) Fibre tract symptoms with progressive paraparesis, diminished sensibility in the legs, and sometimes bladder dysfunction.

Signs:

Diminished or absent tendon reflexes depending on the nerve root or segment involved.

Spastic paraparesis if fibre tracts are involved with a positive Babinski sign.

Muscular atrophy, cutaneous sensibility diminished or lost.

Impaired proprioceptive sensibility. Positive findings at cervical myelogram.

Extraspinal lesions

1. Cervical spondylosis

Description: The disease is due to the degenerative changes in the cervical spine, which begin in the intervertebral discs and affect the posterior intervertebral facet joints secondarily with formation of osteophytes.

Symptoms: In cervical spondylosis, two different kinds of pain may be present:

- a) A deep boring and unpleasant sensation or chronic aching pain of more proximal location with minimal or no dysfunction, localized in the neck, shoulder, scapula, and arm region. Contraction of the cervical muscles elicits pain in the neck, radiating between the shoulders and to the tops of the shoulders with little evidence of nerve root distribution. The pain often radiates to the occiput and into the upper parts of the arms and is aggravated by movements. Typically, the neck is stiff in the morning but softens at use. There is often crepitus and grating on movement. This pain is probably elicited by compression of the ventral motor nerve root.
- b) The second kind of pain is often of a “lightning” character or described as “electric shocks” radiating through the whole length of the extremity, along the distribution of the nerve root. This pain is probably elicited by compression of the dorsal sensory nerve root. Characteristically, the patient is awakened by the pain and frequently also experiences paraesthesias and numbness.

Signs: Localization of pain and tenderness of spinous processes and deep muscles.
Often crepitus and grating on movement.
The pain is aggravated by movements of the neck.
There is no correlation between the degree of pain in the neck and the degree of degenerative changes found on X-ray.
Diminished or absent tendon reflexes depending on the nerve root involved.

2. Brachial plexus injury

Description: Overstretching of the brachial plexus.

Symptoms: Upper plexus lesions result in paralysis, chiefly of the abductors and lateral rotators of the shoulder and flexors of the elbow (Erb type of paralysis). In lower plexus lesions, motor and sensory paralysis occur. (Klumpke type of paralysis).

Signs: Paralysis of the muscles supplied by the brachial plexus.
Clinical history of violent trauma.
Characteristic finding on EMG.

3. Other compression in the thoracic outlet.

Description: Bronchogenic carcinoma of the apex of the lung, i.e. Pancoast's tumour.

Symptoms: This tumour is very rare, and may cause pressure on the vessels and brachial plexus with atrophy of the muscles in the hand and pain around the shoulder and down the arm. Interference with brachial plexus may cause severe pain along the course of the affected nerve. Horner's syndrome may occur because of compression of the cervical sympathetics.

Signs: Clubbing of the fingers is the most frequent peripheral sign of bronchogenic carcinoma.
Pulmonary X-ray.

4. Shoulder-girdle neuritis

Description: Localized neuritis of one or more nerves innervating the shoulder girdle muscles.

Symptoms: Intense pain for several days in the shoulder region followed by paresis of the shoulder girdle.

Signs: Muscular atrophy and weakness of the shoulder-girdle muscles, sometimes bilaterally.

Peripheral nerve entrapments

Median nerve

1. Carpal tunnel syndrome

Description: This is a pure neuropathy with compression of the median nerve in the carpal tunnel at the wrist.

Symptoms: Sometimes, an intermittent aching sensation in the median nerve distribution of the volar aspect of the forearm and wrist, often radiating up the arm into the shoulder with pain and paraesthesias in the thumb, index, long fingers, and even the ring fingers. The symptoms are usually unilateral and are aggravated by prolonged grasping of the hand or pinching with the thumb, index, and long fingers, such as when writing or sewing. Typically, the symptoms occur during the night or early morning hours, awakening the patient. Clumsiness is frequently noted. This condition improves during the day.

Signs: Positive Tinel sign, i.e. reproduction of symptoms at percussion over the median nerve at the wrist.
Positive Phalen sign (Phalen, 1970), i.e. reproduction of symptoms either at maximal flexion or extension of the wrist.
Characteristic EMG findings.

2. Pronator syndrome

Description: In the elbow region, the median nerve can be knicked against the fibrous edge of the flexor digitorum sublimis muscle, or lifted up by the ulnar head of the pronator teres muscle.

Symptoms: Pain and a burning sensation in the palm and first three radial fingers.

Signs: Pronation against resistance with a clenched fist increases the pain. Diminished sensibility is present in the palm and first three fingers, and pressure over the pronator muscle elicits a characteristic pain distribution.

Many patients with the carpal tunnel syndrome experience tenderness over the pronator muscle, but pressure does not cause distal pain radiation.

Ulnar nerve

1. Friction neuritis in the elbow region

Description: The nerve has a superficial position in the ulnar groove at the elbow and is subjected to direct trauma in this region.

Symptoms: Numbness and tingling in the ulnar distribution of the hand and forearm as well as clumsiness. Sensory loss along the ulnar border of the hand, wasting, and weakness of the ulnar-innervated small muscles may be present.

Signs: Tapping and pressure over the ulnar nerve in the ulnar groove reproduce the usual symptoms.
Characteristic EMG findings.

2. Entrapment in Guyon's canal

Description: The nerve is compressed by the volar carpal ligament roofing Guyon's canal, i.e. the shallow trough formed by the pisiform bone and the hook of the hamate bone.

Symptoms: Burning sensation or numbness in the fourth and fifth fingers, weakness, and clumsiness. Decreased sensation along the ulnar border of the hand.

Signs: Tapping and pressure over the nerve at the wrist elicit usual symptoms.
Normal sensibility of the ulnar border on the dorsal aspect of the hand.
Characteristic EMG findings.

Radial nerve

Entrapment of the radial nerve at the elbow region

Description: The deep radial nerve may be entrapped as it passes the fibrous

edge of the extensor carpi radialis brevis muscle insertion or the fibrous slit in the supinator muscle in the elbow region.

Symptoms: Often, sharply localized pain at the lateral epicondyle region. This is often misjudged and treated as epicondylitis. No sensory disturbances.

In some rare cases, the superficial sensory branch of the radial nerve is entrapped when penetrating the proximal portion of the extensor carpi radialis brevis muscle. These patients will experience pain and a depressed sensation over the radial part of the wrist at the “snuff box” region.

Signs: Localized tenderness over the radial nerve at the elbow with reproduction of symptoms at percussion and compression. Supination against resistance reproduces and aggravates the symptoms. Extension of the third finger against resistance reproduces and aggravates the symptoms because the extensor radialis brevis muscle tightens and the compression of the fibrous edge of the muscle on the nerve increases.

Suprascapular nerve

Suprascapular nerve entrapment

Description: This nerve may be entrapped as it passes through the suprascapular notch of the scapula.

Symptoms: Pain localized at the lateral and posterior aspects of the shoulder. The pain is deep and diffusely localized and often radiates down along the outer side to the elbow. Pain is increased by shoulder and scapular motions. In the acute stage, the pain is severe at rest. In chronic entrapment, there is atrophy of the supraspinatus and infraspinatus muscles.

Signs: There is no tenderness in the shoulder joint. Pressure over the suprascapular notch as well as motions of the scapula are painful. Adducting the extended arm passively across the midline of the body is extremely painful, i.e. the cross body adduction test. Suprascapular nerve block of diagnostic importance.

Dorsal scapular nerve

Entrapment of the dorsal scapular nerve

Description: The nerve may be compressed in the distal part of the medial scalene muscle.

Symptoms: Pain along the medial border of the scapula. The pain is often diffusely localized, radiating down the lateral surface of the arm and forearm. In the chronic stage, there is atrophy of the rhomboid muscles and weakness manifested by scapular winging, but not as pronounced as the serratus winging.

Signs: Rhomboid muscle is tender at palpation.
Localized tenderness at the distal part of the medial scalene muscle eliciting typical radiation of pain.

II. Myogenic origin

Myotendinitis

Description: Irritation at the periosteal attachment sites of the neck muscles. Most neck muscles attach at the bone by myofascial tissue that blends into the periosteum. Muscle traction may cause pain and tissue tenderness. Sustained muscle contraction, or overstretching of the muscles, i.e. working in an awkward position for long periods, will cause traction and irritation at the insertion site of the muscle, with myalgic pain and tenderness in the muscle.

Symptoms: Pain and tenderness at the base of the skull radiating down the neck to the shoulders between the scapulae, sometimes radiating down the outside of the arm.

Signs: Often localized tenderness at the insertions of the muscles. By testing the movements of the arm that elicit pain at resistance, the affected muscle can be located:

Abduction: Deltoid and supraspinatus muscles;

Lateral rotation: Infraspinatus and teres minor muscles;

Medial rotation: Subscapularis, latissimus dorsi, pectoralis major, and teres major muscles;

Adduction: Latissimus dorsi, pectoralis major, and teres major muscles;

Flexion of elbow: Long head of biceps muscle;

Extension of elbow: Triceps muscle.

Muscular diseases

Description: Various muscle diseases of the striated muscle of unknown etiology, with widespread destruction of muscle fibres. May develop at any age and in either sex.

Symptoms: Progressive symmetrical weakness of the limb-girdle muscles. The pharyngeal and the laryngeal muscles may be involved.

Signs: Symmetrical atrophies of the limb-girdle muscles.
Myopathic EMG.

Painful arc syndrome

Description: May be due to:

- a) injury of the greater tuberosity
- b) minor tear of the supraspinatus tendon
- c) supraspinatus tendinitis
- d) calcified deposit in the supraspinatus tendon
- e) subacromial bursitis

The pain is caused by mechanical nipping of the tender structure between the tuberosity of the humerus and the acromial process.

Symptoms: Characteristically, the pain is absent or minimal with the arm dependent. During abduction, pain begins at 45° and persists to 160°. Then, the pain lessens or even disappears. Pain is again experienced during the middle arc of the range when the arm is descended from full elevation.

Signs: Localized tenderness between the greater tuberosity and the acromial process.

The typical painful arc, with no pain at full elevation or at rest.

III. Miscellaneous

Causalgia

Description: A posttraumatic syndrome resulting from partial interruption of sensory nerve fibres.

Symptoms: Persistent, severe, diffuse burning pain, almost always in an extremity, triggered by many stimuli.

Signs: Presence of previous trauma, gradually increasing pain in the extremity, the pain becoming severe and unrelenting. The pain is not restricted to one dermatome.

The cutaneous dysaesthesias may be so pronounced that even minimal stimuli may be intolerable.

Vasomotor dysfunction is frequently present with discoloration, hyperhidrosis, and coldness.

Angina pectoris

Symptoms: The pain is often felt over the left thorax radiating to the left arm, but pain can be felt in both arms. The pain is sometimes of paroxysmal character. Typically, the pain is provoked by effort and occurs while the effort is being made and not afterwards.

Signs: The pain is felt on the inside of the arm, often radiating to the elbow and sometimes to the fingers. Negative elevated arm stress test.

ECG at rest and during exercise may be helpful.

Treatment

Some patients with mild TOS will have spontaneous relief and no treatment is indicated. Sometimes, advising the patients to avoid provoking situations is all that is needed, see Fig. 11.

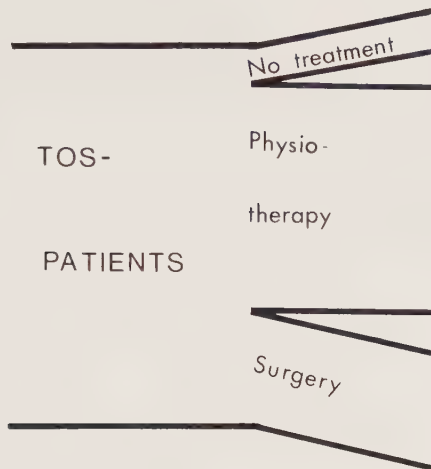


Fig. 11. Management of TOS.

Physiotherapy

In a number of studies, between 50 and 90 % of the patients have experienced relief following physiotherapy (Raaf, 1955; Peet *et al.*, 1956; Nelson, 1957; Rosati & Lord, 1961; Britt, 1967; Urschel & Razzuk, 1972; Dale & Lewis, 1975; Cailliet, 1977; Stallworth *et al.*, 1977, Bakker, 1979; McGough *et al.*, 1979; Crawford, 1980; Dale, 1982). In only one study, however, (Bakker, 1979) the treatment was related to the severity of the symptoms. The patients have been treated with massage, hot packs, ultrasound and, in most studies, strengthening exercises of the muscles of the shoulder girdle. In some instances, cervical traction has been used to differentiate between TOS and cervical spondylosis, as such traction is known to aggravate the symptoms in patients with TOS (Dale & Lewis, 1975).

Surgical treatment

Since Coote first resected a cervical rib in 1861, there has been a number of surgical approaches reflecting the changing perceptions of the causative mechanism of compression in the thoracic outlet (Coote, 1861). In 1905, Murphy stated that the first rib compressed the neurovascular structures in the thoracic outlet and recommended resection of the rib (Murphy, 1905). In 1927, when Adson and Coffey published their paper on the anterior scalene muscle as the compressing mechanism, the era of scalenotomy was started (Adson & Coffey, 1927). For many years, scalenotomy was considered the best treatment of compression syn-

dromes in the thoracic outlet (Craig & Knepper, 1937; Adson, 1947; Gage & Parnell, 1947). The significance of the first rib as the common denominator of the compression syndromes in the thoracic outlet was revived by Clagett 1962 as the failure rate of scalenotomy was too high (Clagett, 1962).

There are various routes to first rib resection, i.e. the transaxillary approach (Dale & Lewis, 1975; McGrougk *et al.*, 1979; Roos, 1966; Roos, 1980a; Roos, 1981), the posterior parascapular thoracoplasty (Martinez, 1979), the anterior supraclavicular approach (Thomas *et al.*, 1978; Thomas, 1979; Hempel *et al.*, 1981), and the infraclavicular approach (Nelson & Davis, 1969; Nelson & Jenson, 1970; Murphy *et al.*, 1980). Some surgeons use a modified transaxillary approach with the patient in a supine position and with his arm elevated.

Summary

A review of the literature reveals that the understanding of TOS has increased although several questions remain to be answered.

The value of laboratory studies on arterial or venous compression as an objective assessment of a concomitant compression of the brachial plexus has not been sufficiently investigated. The incidence of TOS and its possible correlation to certain occupations is not known. Nor do we know which TOS patients will benefit most from physiotherapy and in which cases surgery is the best therapy.

Aims of the study

The aims of the present investigation have been

- to assess the effect of Doppler flowmetry in the diagnosis of TOS (I)
- to establish possible correlations between occupations implying repetitive straining of the arms and the presence of compression symptoms in the thoracic outlet (II)
- to assess the incidence of these symptoms in normal subjects (I)
- to assess the effect of correcting postural disorders by physiotherapy in TOS patients (III)
- to assess the effect of surgical treatment of patients with TOS (IV).

Chapter two

Present investigations

I. Non-invasive investigation of vascular compression in patients with thoracic outlet syndrome

Three hundred and seventy-seven patients with brachial pain, suggestive of the thoracic outlet syndrome, and 63 controls were examined clinically and by means of Doppler flowmetry. A 10 MHz ultrasonic Doppler flow detector was used to detect diminished flow in the radial artery with the arms in different provocative positions.

Results

Two hundred and thirty-seven patients (63 %) had compression symptoms of the brachial plexus, while 11 patients (3 %) had symptoms of isolated vascular compression. One hundred and twenty-nine patients (34 %) had symptoms not related to the thoracic outlet syndrome. The incidence of TOS symptoms in the unselected controls was 9,5 %.

A significant correlation, $p < 0.001$, was established between concomitant compression of the brachial plexus and compression of the subclavian artery in the thoracic outlet in the patients, see Fig 12, determined by the non-invasive Doppler flow test. The specificity was 53 % and the sensitivity 74 %. In 172 patients, we made a comparison between arterial compression symptoms assessed by palpation and Doppler flowmetry. There was a significant correlation, $p < 0.001$, with a specificity of 63 % and a sensitivity of 96 %. Total obstruction of the radial pulse with the arms in provocative positions in the control subjects was present in 17 %.

Comments

Because of the close anatomic proximity between the subclavian artery and the brachial plexus, concomitant compression of both structures in TOS patients is significantly correlated. Even in normal subjects, however, arterial compression can be provoked without concomitant neurological symptoms. The Doppler flowmetry technique adopted in this study gave a number of false positive and negative results. Arterial compression can easily be assessed by palpation and auscultation. In rare cases with suspected vascular lesions indicated by signs of distal embolization, objective methods are useful.

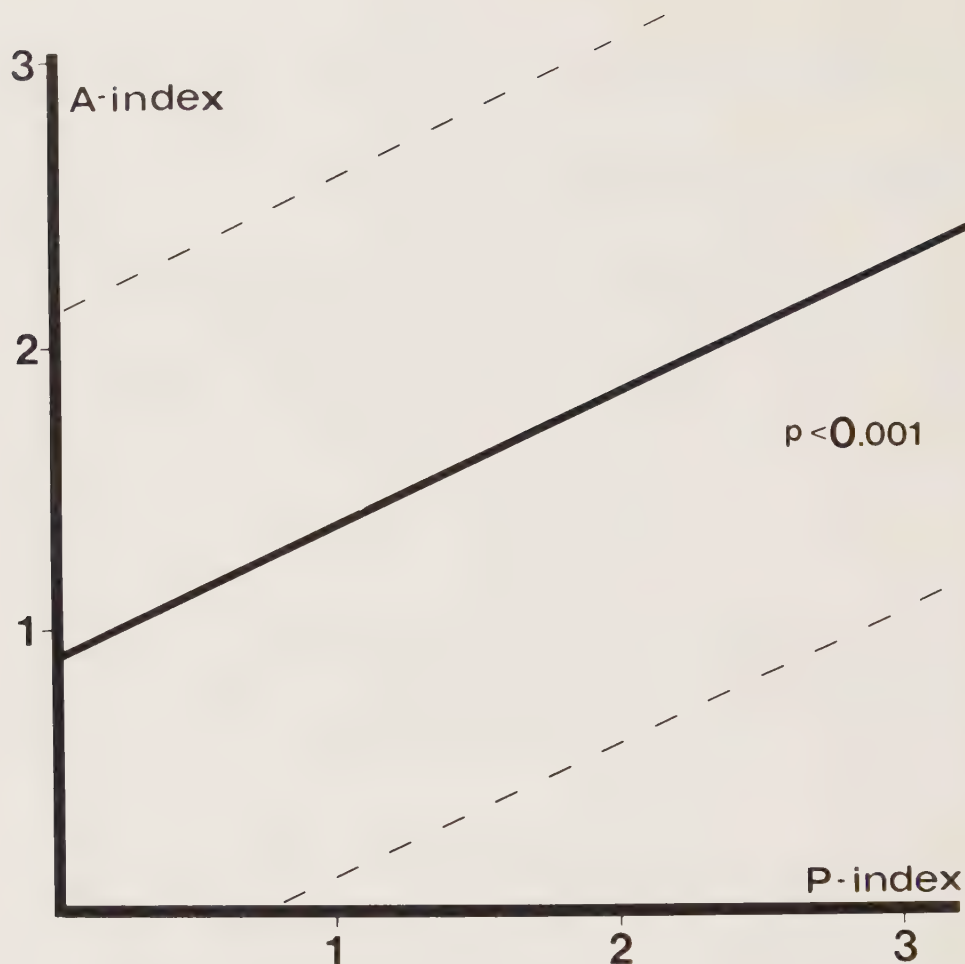


Fig. 12. Correlation between arterial (A-index) and plexus (P-index) compression in patients with TOS.

II. Cervicobrachial disorders in certain occupations with special reference to compression symptoms in the thoracic outlet

One hundred and ninety-one workers from three different vocational groups were examined with reference to prevalence of symptoms from the upper extremities. Ninety-two industrial workers, 62 office workers, and 37 cash register operators were included in the study. The industrial work consisted of welding, drilling, turning, and assembly-line work. The office workers typed and edited technical texts using word processing keyboards and display units. The cash register

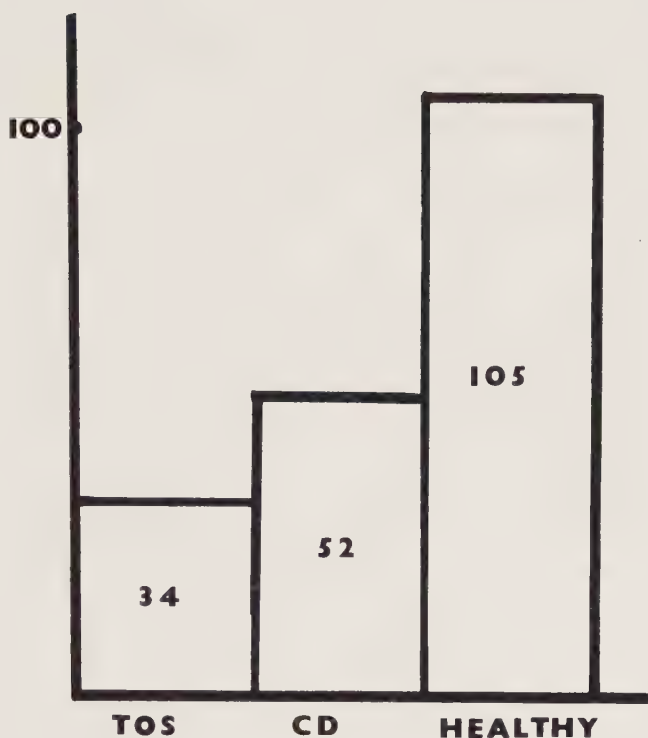


Fig. 13. Total material of workers differentiated into 3 groups according to presence of symptoms in the cervicobrachial region. CD = cervicobrachial disorders not due to TOS.

operators' work forced them to keep their right arms in half abduction and almost 50° elevation from the bodies. At the same time, they twisted their bodies lifting articles with their left arms. According to pre-determined criteria, the workers were divided into three groups, i.e. those without any cervicobrachial symptoms, those with compression symptoms in the thoracic outlet, and those with other cervicobrachial symptoms, see Fig 13.

Results

Fifty-five percent (105/191) of the workers had no symptoms. Symptoms in the cervicobrachial region occurred in 45 % (86/191) of all workers with a female predominance of almost 2:1. Among these workers symptoms of brachial plexus compression could be provoked in 34, i.e. 18 % of all workers. Pronounced symptoms of brachial plexus compression were present in only 2 % (4/191), however. In workers with symptoms of brachial plexus compression, the female predominance was almost 3:1.

The incidence of cervicobrachial symptoms not due to TOS was highest among the heavy industry workers, 29 % (27/92), and among the cash register operators, 43 % (16/37). The same incidence pattern in the two worker groups applied to brachial plexus compression symptoms.

Comments

Other studies have shown a high incidence of cervicobrachial symptoms in certain occupations (Nishiyama *et al.*, 1973, Duncan & Ferguson, 1974; Maeda, 1975; Ohara *et al.*, 1976a; Ohara *et al.*, 1976b; Onishi *et al.*, 1976; Petersen *et al.*, 1976). This has been explained in terms of heavy workloads in awkward work postures, repetitive and monotonous work, or unnatural static positions of the arms with continuous muscle tension in the shoulder girdle muscles.

Our study has shown an unexpectedly high incidence of brachial plexus compression symptoms in certain occupational groups. As the incidence pattern coincided with that of the cervicobrachial symptoms, the explanations are probably the same as mentioned above. Heavy work with the arms seems to provoke symptoms to a high degree, but the work position and continuous muscle tension, as in the cash register operators, provoke symptoms even more frequently. The high incidence of myotendinitis caused by severe strain of the shoulder girdle muscles in heavy industry workers may elicit compression symptoms in the thoracic outlet in susceptible workers due to irritation of the plexus. This leads to an inappropriate stimulation of the nerves, eliciting muscle spasms and further irritation of the plexus and a vicious circle is established. In the cash register operators, the combination of keeping the arm abducted and the continuous muscle tension constricts the thoracic outlet. The muscle tension may elicit a vicious circle in susceptible workers. These concurrent factors seem to be the reason why the incidence of brachial plexus compression symptoms is highest in this group of workers.

III. Physiotherapy in patients with thoracic outlet syndrome

Ninety-nine patients with TOS symptoms, with a female predominance of 2:1, have been treated with physiotherapy. In all patients, the pelvis was tilted backwards and the lumbar lordosis was diminished or even substituted by a slight kyphosis. The thoracic kyphosis was diminished and at the transition to the cervical spine there was often a marked kyphosis. The scapulae were protracted and the hips and knees hyperextended in the standing position. Movements in the shoulder joints were impaired. All TOS patients were treated in accordance with a new concept of correcting postural disorders.

The treatment consisted of reposition of the sacro-iliac joint, posture training, coordination exercises, ergonomic instructions, and treatment of muscular pain.

The aim of the physiotherapy was to regain a normal posture and muscle range, leading to a relief of the pain in the thoracic outlet. The different exercises are closely related, i.e. posture training is made possible after reposition of the sacro-iliac joint. The posture training enables the patient to make the best use of the coordination exercises, which in turn makes it possible to treat the painful muscles.

The patient is taught how to find the vertical alignment of the body (i.e. the line from the ear through the shoulder, hip and in front of the lateral malleol) by reposition of the pelvis. The patient is then instructed to do these posture exercises in front of a mirror at home. The gait coordination is trained in different positions, i.e. standing, walking, sitting, and lying. The coordination exercises are made more effective by the posture training and are a part of the treatment of the painful muscles. Specific treatment of muscle pain cannot start before the coordination exercises are well under way. A common rule in the treatment of muscle pain is stretching of the antagonistic muscle, i.e. stretching of the triceps muscle in biceps tendinitis, stretching of the psoas muscle in painful shortening of the piriformis muscle, stretching of the adductor muscles in contracted and spastic abductors in the pelvic region. Tendinitis of the pectoralis minor muscle, however, is treated by stretching the muscle itself.

Finally, ergonomic instructions are given to the patient with the common aim of preventing the pelvis from being locked in a backward tilted position. The patient is instructed not to work with the body bent forward for long periods. In standing work positions, the patient is told to stand 15 to 20 cm away from the table and to rest his pelvis against the edge of the table. In sitting work positions, the patient is instructed to sit either on the edge of the chair with his feet under the chair or sit on a wedge-shaped pillow, keeping the pelvis tilted forward.

Results

Complete relief was obtained in 26 % (26) and in 30 % (30) there was a marked improvement. In 5 % (5) there was only a slight improvement and in 38 % (38) no relief of the symptoms could be demonstrated. In patients with mild to moderate symptoms, the result of the therapy was good in 83 % and 77 %, respectively. If the symptoms were severe and were elicited by trauma, the results of the physiotherapy was poor.

Comments

In almost every third patient who obtained complete relief, the symptoms could be provoked by the elevated arm stress test (Roos' test). This indicates that the postural disorders can not be the primary cause of the thoracic outlet syn-

drome, but rather act as an eliciting factor in susceptible people, i.e. persons with congenital bands as predisposing factors. Forty patients had previously been treated with ultrasound, massage, hot packs, or strengthening exercises of the shoulder girdle muscles with no relief of their symptoms. They obtained improvements to the same degree as the remaining group of patients with the new concept of physiotherapy. The results obtained in the 40 previously treated patients indicate that this new concept of physiotherapy can well defend its place in the therapeutic arsenal.

IV. Surgical treatment of the thoracic outlet syndrome

Four-hundred and ten patients were examined for suspected thoracic outlet syndrome. Sixty-three (15 %) patients with severe TOS symptoms with no benefit of conservative treatment were operated, 9 bilaterally, making a total of 72 operations. Four patients were operated because of vascular symptoms, i.e. three had deep venous thrombosis, and one patient had a pronounced arterial insufficiency.

All except two patients were operated by the transaxillary approach.

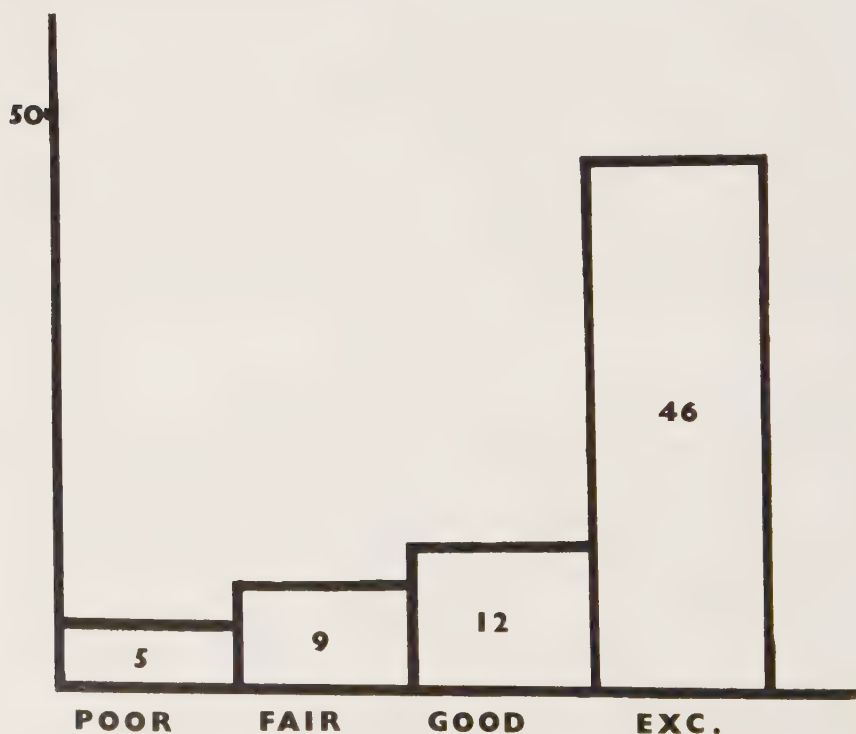


Fig. 14. Clinical evaluation of operative treatment in 63 patients (9 operated bilateral making a total of 72 operations).

Results

Excellent results were obtained in 64 %. In 17 %, only minor symptoms in the cervicobrachial area persisted, see Fig 14. In 9 patients (12 %), only partial relief was obtained and 5 patients (7 %) had poor results.

Comments

Good or excellent results have been obtained in 57 % to more than 90 % by means of surgical decompression of the thoracic outlet (Lo-A-Njoe, 1974; Dale & Lewis, 1975; Dunant, 1976; McGough, Pearce & Byrne, 1979; Roos, 1979). One of the main reasons for the varying degree of success by surgical treatment is incorrect diagnosis.

As the results of surgery are not optimal, the indications for surgery must be strict, surgical decompression being the last resort. However, patients with severe and disabling symptoms respond poorly to physiotherapy. The transaxillary approach seems to offer the best possibilities for total decompression of the thoracic outlet.

Chapter three

General discussion

Patients with symptoms in the neck, shoulder or arm regions constitute a large and mixed group. The causes of their symptoms are multiple and many patients have had their symptoms for long periods of time. Due to the prolonged symptom periods, many of the patients develop psychosomatic symptoms not related to TOS. Often, the patients have consulted a number of doctors without getting any help. At a brief medical examination, the psychosomatic symptoms will predominate and the real cause of the patient's symptoms will not be revealed. Diagnosing TOS is a matter of weighing together the detailed history and examination of the patient. This is always a time-consuming task, but some important symptoms and signs can aid the diagnosis. Pain in the shoulder, arm, and hand is invariably present in almost all TOS patient. The pain is characteristically distributed along the ulnar aspect of the arm and appears after, rather than during, provocation. Paraesthesias and numbness along the ulnar aspect of the forearm to the 4th and 5th fingers are almost as common. Weakness and fatigue of the affected extremity and nocturnal pain and paraesthesias are also usually present. A detailed history is of utmost importance in the diagnosis of TOS.

At clinical examination, reproducing the symptoms in the elevated arm stress test (Roos, 1976) is one of the cornerstones in the diagnosis. Palpation for tenderness over the brachial plexus and reproduction of pain and paraesthesias in the arm and hand at tapping in the supraclavicular fossa are other important tests. Diminished sensibility in the ulnar aspect of the hand and forearm as well as weakness and early fatigue of the arm further strengthen the reliability of the diagnosis of TOS. There are a number of tests to assess vascular and neurologic compression in the thoracic outlet.

Vascular compression can be assessed by non-invasive methods or by arteriography. The Doppler flowmetry adopted in our study (I) is an easy technique of assessing arterial flow obstruction. Arterial compression commonly occurs also in normal subjects at provocation, however (Wright, 1945; Raaf, 1955; Gilroy and Meyer, 1963; Torriani, 1971; Dale & Lewis, 1975; Rainer & Sadler, 1975; Roos, 1976; Gergoudis & Barnes, 1980; I). Even if arterial compression is significantly correlated to brachial plexus compression, arterial compression is not synonymous with TOS. However, clinical vascular tests, i.e. pulse palpation and infraclavicular auscultation, should be used routinely. Thus occlusion at the Roos maneuver *and* concomitant neurologic symptoms strengthen the diagnosis of TOS. At the same time, it is important not to rely too much on vascular tests as the main diagnostic means. Arterial compression causing arterial occlusion or aneurysmal formation with distal embolization is a rare phenomenon, i.e. 1/2–2 % of all TOS patients, but constitutes an indication for arteriography (Roos, 1976; I;

IV). Reports of a higher incidence of vascular compression in the TOS patients probably reflects either a specific referral pattern or an exaggeration of the importance of vascular compression in the diagnosis of TOS (Dale, 1982). In the rare cases of arterial injury with occlusion or distal embolization, however, early diagnosis with arteriography is important for the viability of the arm.

Neurologic compression can be assessed either by measuring the nerve conduction velocity of the ulnar nerve (Urschel & Razzuk, 1972; Gilliat, 1976; Stanton *et al.*, 1978; Glassenberg, 1981) or by measuring the sensory action potentials of the ulnar nerve (Gilliat *et al.*, 1978), as the main neurologic symptoms are sensory rather than motor disturbances. Other neurophysiologic methods determine evoked potentials or an increased F wave latency in patients with suspected TOS (Wulff & Gilliat, 1979; Glover *et al.*, 1981). Neither one of these methods is decisive in all cases of brachial plexus compression, however. Pathologic neurophysiologic readings are present only in patients with evident atrophy of the intrinsic muscles of the hand. The readings are normal in patients without atrophies and even in patients with disabling pain and paraesthesias (Gilliat *et al.*, 1978; Wulff & Gilliat, 1979; Glassenberg, 1981). Neurophysiologic tests are very useful in diagnosing peripheral nerve entrapments, however, (Jebsen, 1967; Caldwell *et al.*, 1971).

A number of factors may elicit TOS. One predisposing factor is cervical ribs (Sanders *et al.*, 1968; Johnson, 1974; Dale & Lewis, 1975; Roos, 1976). Cervical ribs occur in the normal population at a rate of 0.06–1 % (Etter, 1944; White *et al.*, 1945; Crimm, 1952). In TOS patients, cervical ribs are present in 1–15 % (Sanders *et al.*, 1968; Johnson, 1974; Dale & Lewis, 1975; Roos, 1976).

In our study (IV), cervical ribs were present in 6.5 % of the patients. The most important predisposing factor, however, seems to be the congenital fibrous or muscular bands that mechanically compress the thoracic outlet (Roos, 1976). In a cadaver study, Roos could demonstrate such bands in 33 % of the bodies (Roos, 1976). In almost one thousand operations for thoracic outlet syndrome, congenital bands were present in 98 % of the patients (Roos, 1976; Roos, 1979). In our series, we looked for congenital bands in the last 30 patients. In 22 of them, i.e. 73 %, different bands were present (IV). Although many people have congenital bands, few develop compression symptoms in the thoracic outlet. Both cervical ribs and congenital anomalous bands may exist in some subjects with no TOS symptoms. Just as apparent is the fact that in some TOS patients, no cervical ribs or congenital anomalies can be visualized. This illustrates the difficulty in making the diagnosis.

Furthermore the presence of cervical ribs or congenital anomalies does not explain all TOS cases. Thus, other factors such as trauma to the head, neck or shoulder may cause TOS (Woods, 1965; Roos & Owens, 1966; Sanders *et al.*, 1968; Dale & Lewis, 1975; Capistrant, 1977; Kelly, 1979; Sanders *et al.*, 1979; Martinez, 1982a). Other eliciting factors are postural disorders (III) or occupational hazards (II). In our study, we found common postural disorders such as backward tilting of the pelvis, diminished lumbar lordosis, and thoracic kyphosis.

In some patients the scapulae were protracted and movements in the shoulder joints diminished or absent (III).

Most often, the occupational hazards seemed to be a combination of abduction and elevation of the arm and continuous muscle tension as seen in cash register operators. Heavy work, including welding, drilling, turning, and assembly-line work also provoked compression symptoms in the thoracic outlet, but not as frequent as in the cash register operators. The work position and extent of continuous tension of the shoulder girdle muscles, rather than the actual heaviness of the work, seemed to elicit compression symptoms of the brachial plexus to a higher degree (II). Postural or occupational factors, however, are secondary factors eliciting TOS in susceptible persons, i.e. persons with congenital bands.

Apparently, the thoracic outlet syndrome is a common disease. In our study of 191 workers, 18 % has compression symptoms in the thoracic outlet at provocation, although only 4 % had pronounced symptoms (II). The incidence is higher in women and, in our study, the female predominance was almost 3:1 (II). The incidence varies depending on the kind of work involved, i.e. it is highest in the cash register operators (32 %) and is only 10 % among the office workers. The incidence is to some degree artificial, however, because the workers were examined at their place of work and only four of them had previously sought medical advice for TOS symptoms.

Informing patients with mild symptoms to avoid provoking positions often is all that is needed. If the symptoms are not prevented in this manner, other measures such as physical therapy must be taken. Hot packs, massage, and ultra-sound are often effective in inflammatory diseases, but often give only a temporary relief of the TOS symptoms (Roos, 1976; III). Good results have been obtained with strengthening exercises of the shoulder girdle muscles (Raaf, 1955; Peet *et al.*, 1956; Nelson, 1957; Rosati & Lord, 1961; Britt, 1967; Urschel & Razzuk, 1972; Dale & Lewis, 1975; Cailliet, 1977; Stallworth *et al.*, 1977; Bakker, 1979; McGough *et al.*, 1979; Dale, 1982). In a number of patients, we tried the exercise program advocated by Britt (Britt, 1967). The results were poor, however, i.e. only 1 of 12 patients had temporary benefits, whereas aggravation of the symptoms occurred in 8 patients. Furthermore, we found that all patients with TOS symptoms had common postural disorders (III). Such disorders as a TOS-eliciting factor were mentioned already by Todd (Todd, 1912; Todd, 1922). The postural disorders were not relieved by Britt's exercise program, and this was probably the cause of the poor results. A completely new approach to correct the postural disorders led to remarkably good results in mild and moderate cases (III). The results were equally good in patients previously treated with hot packs, ultra-sound, massage, or by Britt's exercise program. The results were poor in patients with severe symptoms, however. This was probably due to the fact that a vicious circle of muscle spasm, pain, and irritation of the brachial plexus had been established by the predisposing congenital bands. In the mild or moderate cases, the circle apparently is reversible which explains the good results of physiotherapy in

these cases. In the severe cases, the vicious circle is irreversible and physiotherapy will not offer any relief, no matter what kind of therapeutic method is used.

Surgical treatment with decompression of the thoracic outlet and excision of all compressive mechanisms is the only acceptable method in patients with physiotherapeutically intractable symptoms (III, IV).

There are several surgical approaches to the thoracic outlet, i.e. the transaxillary (Roos, 1966; Urschel & Razzuk, 1972; Dale & Lewis, 1975; McGough *et al.*, 1979; Roos, 1980a; Roos, 1981) the posterior parascapular (Martinez, 1979), the supraclavicular (Thomas *et al.*, 1978; Thomas, 1979; Hempel *et al.*, 1981), the infraclavicular (Nelson & Davis, 1969; Nelson & Jenson, 1970; Murphy *et al.*, 1980) and the modified transaxillary approach with the patient in a supine position with his arm elevated. For a total decompression of the thoracic outlet, it is mandatory to resect the rib all the way to the transverse process. Secondly, excision of all congenital bands in the outlet area is mandatory to get a total decompression. The supraclavicular approach is a very difficult and potentially dangerous dissection around the brachial plexus and the vessels covering the rib. In the infraclavicular route, only the anterior two thirds of the rib can be removed safely. Thus, total decompression of the thoracic outlet is not possible by these approaches. With the dorsal parascapular approach, the surgeon can make a total decompression. This route differs from the transaxillary only insofar that there is a higher postoperative morbidity due to the thoracoplasty. Although the operative field is deep and tunnelshaped in the transaxillary approach, it offers the best possibilities for complete resection of the first rib and congenital bands. The neurovascular structures are located *behind* the rib, outside the operative field, as opposed to the supraclavicular approach. In the 90° thoracotomy position advocated by Roos, (Roos, 1966), the surgeon can look straight into the operative field, as opposed to the modified supine position where the surgeon will have trouble getting the light into the field which makes the operation unnecessarily difficult.

As the results of first rib resection are not optimal, other surgical approaches have been tried. Recently, scalenectomy has been recommended by some authors as the best treatment of TOS (Stallworth *et al.*, 1977; Sanders *et al.*, 1979). Stallworth *et al.* reported good results by transaxillary division of the pectoralis minor muscle, scalenus anterior, or subclavian muscles (Stallworth *et al.*, 1977). Sanders *et al.* compared supraclavicular ectomy of the anterior and middle scalene muscles with first rib resection. The results were equally good in both groups (Sanders *et al.*, 1979). Sanders *et al.* state that, in neck trauma, the sudden onset of symptoms must be due to irritation of the scalene muscles, and that scalenectomy of *both* the anterior *and* the middle scalenes is an adequate treatment (Sanders *et al.* 1979). The sudden onset after trauma may also be explained by muscle trauma with localized hemorrhage or swelling, causing muscle spasms that constrict the thoracic outlet. In susceptible persons, this activates the underlying congenital anomaly leading to a compression of the brachial plexus. This elicits

inappropriate stimuli which further increase the muscle spasm and a vicious circle is established. In 30 % of the anatomical dissections, Gage & Parnell found that the *upper* cord of the brachial plexus passed *through* the anterior scalene muscle (Gage & Parnell, 1947). This is an important point, as scalenectomy offers the best possibilities in patients with compression of the *upper* plexus cord (Roos, 1979, 1982). The anterior scalene muscle is not associated with the lower plexus cord where most of the TOS symptoms originate, however. The middle scalene muscle, on the other hand, is imminently associated with the brachial plexus. Through the supraclavicular approach, the middle scalene muscle and the congenital bands usually present in TOS patients are very difficult and dangerous to remove.

Secondly, the patient is put in a worse position due to scar tissue formation in the thoracic outlet should the scalenectomy fail. This makes the second operation far more difficult than the first. Several surgeons have cautioned against early optimism in scalenectomy and have pointed to the high recurrence rate after several years following scalenectomy (Campbell, 1977; Roos, 1977b; Urschel, 1977; Razzuk, 1979).

Initially, we used the transaxillary approach with the patient in a supine position. The operation was difficult because of poor accessibility. Changing to the 90° thoracotomy position advocated by Roos (Roos, 1966; Roos, 1980a; Roos, 1981), the accessibility was remarkably improved, making the operation easier. We chose the transaxillary approach because we considered this the best way to treat all the causative mechanisms in the thoracic outlet. Total decompression of the thoracic outlet cannot be achieved by the supraclavicular or infraclavicular approaches. The posterior parascapular approach offers the same possibility as the transaxillary approach. The main objection to the posterior approach, however, is the higher postoperative morbidity. Furthermore, we did not consider it necessary to perform a thoracotomy to relieve the thoracic outlet when it can be done transaxillary and extrapleurally with a short hospitalization. The operative results vary and in many studies they are not optimal (Dale, 1982). Thus, there are still a number of patients who have not been helped by the operations. The explanation of the poor results may be incorrect diagnosis, and this is probably the most important reason. Other reasons may be insufficient surgical decompression of the thoracic outlet or permanent nerve injury. Another reason is regeneration of the first rib.

The recurrence rate after first rib resection varies between less than 1 % to about 15 % (Urschel & Razzuk, 1972; Roos, 1976; Stallworth *et al.*, 1977; Roos, 1979; Sanders *et al.*, 1979; Dale, 1982).

In our study, the rate was 5 % with a median observation time of 2 1/2 years (IV). The early recurrences are probably due to scar tissue formation involving the brachial plexus. Usually, a trauma to the head, neck or shoulder regions elicits the symptoms in late recurrences. The trauma probably causes a hemorrhage in

the muscle with subsequent scar tissue entrapping the plexus. There are two distinct patterns of recurrent TOS (Roos, 1980b).

Type I is caused by postoperative scar tissue fixation of the lower trunk of the brachial plexus, i.e. the C:8 and Th:1 nerves. The nerves are fixed in the rigid scar tissue at the place of the removed first rib, causing compression symptoms of the lower trunk with principally ulnar symptoms.

The Type II pattern involves the *upper* trunks of the brachial plexus, i.e. the C:5–C:6 och C:7 nerves, that are fixed in scar tissue caused by reattachment of the anterior scalene muscle to the cupola of the lung, the subclavian artery, or even to the nerves themselves (Roos, 1980b). The symptoms are those of upper plexus compression, i.e. pain in the neck radiating back into the rhomboid area and to the shoulder, down the outside of the arm to elbow level. The cervical muscles are in painful spasm causing hemicranial headache and pain at the base of the skull.

Initially, the symptoms of Type I or II recurrences are mild and intermittent, gradually becoming worse with unrelenting pain. In most cases, surgical neurolysis of the entrapped nerves is the only possibility offering relief of the symptoms. The surgical approach depends on the type of recurrence, i.e. in the Type I recurrence, neurolysis is done by the transaxillary route to release the lower trunk from the scar tissue. In the Type II recurrence with upper plexus involvement, the release is accomplished by the supraclavicular approach. However, prevention of recurrent TOS is extremely important as it minimizes excessive scar tissue formation following first rib resection. First, the rib must be removed all the way up to the transverse process to prevent the plexus from hooking over the stump. Secondly, the anterior scalene muscle must be pulled down and cut as high as possible to prevent the muscle from reattaching, taking care not to injure the subclavian vessels or the phrenic nerve in this procedure.

Conclusions

Diagnosing TOS is a difficult undertaking and, so far, no objective methods superior to a detailed history and examination of the patient are available. We have investigated the arterial compression by means of the Doppler flowmetry technique (I). Obstruction of flow was easy to register and the arterial compression was significantly, $p < 0.001$, correlated to concomitant compression of the brachial plexus. In spite of this, the Doppler flowmetry is of little help in the diagnosis of TOS, since arterial compression often occurs at provocation even in normal subjects.

Continuous muscle tension and adverse positions with the arms, i.e. prolonged elevation and abduction seem to elicit TOS symptoms in cash register operators (II). Heavy work, such as welding, drilling, turning, or assembly-line work cause an overdue exertion of the shoulder girdle muscles which seems to elicit TOS symptoms in susceptible workers.

The incidence of TOS was unexpectedly high, or 18 % of the 191 examined workers (II). In most cases, however, the symptoms were mild and only 4 % of the workers has sought medical advice due to symptoms compatible with compression in the thoracic outlet. The incidence of compression of the brachial plexus symptoms was highest among the cash register operators (32 %). This incidence was almost twice as high as among the workers in heavy industry and more than three times the incidence among the office workers. In the 63 control subjects, symptoms of thoracic outlet compression could be provoked in 9.5 %, although no one had sought medical advice. The female predominance in this group was 2.4:1.

Correction of the postural disorders gave excellent results in the majority of patients with mild or moderate TOS symptoms (III). Few patients with severe symptoms or TOS caused by trauma, obtained any relief. Since physiotherapy is harmless and easy to administer, and since patients with mild to moderate symptoms constitute the majority of TOS patients, it should be tried in all patients with TOS. If the symptoms are aggravated or no relief is obtained in two or three months, however, surgery should be considered.

In patients with severe symptoms not helped by physiotherapy, surgical decompression of the thoracic outlet is the best treatment (IV). The degree of success in surgery varies, however, indicating that great care should be taken in diagnosing TOS, surgery being indicated only when physical therapy has failed to alleviate the symptoms after 2–3 months of treatment.

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Chapter four

I

Non-invasive investigation of vascular compression in patients with thoracic outlet syndrome

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Summary. Three hundred and seventy-seven patients with brachial pain and 63 controls have been examined regarding presence of symptomatic compression of the brachial plexus and subclavian vessels (Thoracic Outlet Syndrome = TOS). Each patient was examined clinically and by Doppler flowmetry. Two hundred and thirty (61 %) had moderate to pronounced compression of the brachial plexus and 11 (2.9 %) isolated compression of the subclavian artery or vein. One hundred and twenty-nine patients had symptoms not related to TOS. The prevalence of TOS was almost twice as common in women as in men, 1.76:1. The TOS patients were younger than the non-TOS patients, $P<0.05$. There was a significant correlation between arterial compression measured by Doppler flowmetry and the presence of compression of the plexus, $P<0.001$, but the validity of this method was not satisfactory, giving both false positive and negative results. Furthermore, there was a very good correlation between arterial compression assessed clinically and by Doppler flowmetry, $P<0.001$. Thus, the diagnosis of TOS is still a clinical judgement, the Doppler technique adding very little and any significant vascular compression being easily detected by clinical assessment.

Introduction

The thoracic outlet syndrome (TOS) is a symptom-complex elicited by compression of the brachial plexus and the adjacent subclavian vessels. The neurovascular structures are mainly compressed between the clavicle and the first rib. The symptoms may be entirely neural, arterial or venous, or a combination of these.

TOS seems to be a common disease and it is important to know the presence and extent of vascular compression because this feature bears a potential risk for permanent vascular damage to the vessel and may consequently lead to emboli or occlusion of the artery or venous thrombosis (Gjöres, Svendler & Todoreskov, 1975). Another aspect is the possible correlation between arterial compression and presence of neurological symptoms since arterial compression can easily be recognized and may therefore serve as a guide to nervous compression, a symptom not easily diagnosed objectively. There are several reports regarding assessment of arterial compression using phonoangiography (Dunant *et al.*, 1974; Hehne, Dunant & Waibel, 1975) and oscillometry (Dunant, 1976; Lo-A-Njoe, 1974), but little has been done regarding quantitative measurements of arterial compression and its correlation to other symptoms. For our study we have adopted a procedure based on Doppler flow determination. This technique allows both qualitative and semi-quantitative measurement of arterial compression elicited by different provocative procedures. A brief presentation of the method has been given previously (Thulesius, 1978)

Since the clinical diagnosis of TOS calls for taking an elaborate history and a very careful examination of the patient, which may be rather cumbersome, this study was carried out in order to see if there was a correlation between arterial and brachial plexus compression and if assessment of arterial compression could be used as a supportive diagnosis in cases with compression of the brachial plexus.

Material and methods

Patients and controls

The patient material consisted of 377 cases admitted to the surgical department during the period of 1976–1979 because of symptoms from the upper extremities suggesting TOS. Moreover, 63 “controls” were examined. These subjects were 30 unselected young soldiers and 33 female nursing students representing a whole class. They were supposed to be free of symptoms and had never applied for medical advice or treatment concerning the upper extremities. Details of the patients and the “controls” are presented in Table 1.

Doppler flowmetry

The principle for detection of arterial obstruction was to record blood flow in the radial artery with a continuous wave 10 MHz ultrasonic Doppler flow detector. Initially we used a directional model (Parks 806 C) and recorded flow tracings, but later we limited the procedure to the audio-signals since diminution and cessation of the flow is easily recognized with this method. For this purpose a simple equipment (Parks 812) is sufficient. Recording from the artery was achieved using a flat-probe strapped to the radial artery at the wrist. Arterial flow was detected in eight standard positions. The positions were as follows: sitting with the arms in the lap; sitting with the arms elevated and abducted with flexed elbows and forearms horizontal with head looking forward—to the right and to the left; sitting with the arms elevated and abducted with flexed elbows and forearms straight up with the head looking forward (AER-position)—to the right and to the left; sitting with the arms elevated straight up. The flow pulsations were evaluated according to a 4-graded scale ranging from 0 to 3, 0 being normal pulses and 3 total cessation of flow with 1 and 2 meaning partial reduction of flow.

Table 1. Total material of patients with symptoms from upper extremities and “controls”. Number of cases (*n*), sex, age (years) and relationship. (TOS=Thoracic Outlet Syndrome, non-TOS=brachial pain, not related to TOS). SD=standard deviation

	<i>n</i>	Women/men	ratio	Age (mean±1 SD)	Range
Total	377	241/136	1,77:1	38,2±11,1	(13–71)
TOS	248	158/90	1,76:1	37,5±10,7	(13–60)
non-TOS	129	83/46	1,80:1	41,1±12,1	(18–71)
“Controls”	63	33/30	1,1:1	19,8±2,9	(18–39)

In addition to recording flow we also performed auscultation with a stethoscope below the clavicle with the arms in the same positions as used for flow measurements. Auscultatory findings were evaluated according to a 4-graded scale ranging

from no bruit: 0, to a marked continuous systolic murmur: 3. The combination of bruits and flow compression ratings may, in pronounced cases result in disappearance of flow signals and no bruit.

Clinical evaluation of nerve and vascular compression

At first a careful patient history was recorded. Current complaints such as pain, tingling, paraesthesias, numbness or early fatigue were listed and mode of provocation (when, where, how?) was noted. It was ascertained if the symptoms were aggravated or elicited by certain activities or movements during work or at home, e.g. roof-painting, typing, window cleaning, vacuum cleaning, knitting or doing the hair. Previous trauma involving the shoulder girdle, e.g. collar bone fracture, and sick-leave due to severe symptoms as well as previous treatment or medication was noted.

The clinical examination started with an assessment of the neuromuscular function of the hands and arms. Each test in the neurologic examination was rated by a 4-graded scale from 0 to 3 according to increasing severity of symptoms, 0 meaning no symptoms, 1 slight, 2 moderate and 3 pronounced symptoms. Atrophy of the intrinsic muscles of the hand and of the thenar and hypothenar region was noted and graded. Muscle strength was tested and graded (excellent, good, fair, poor) in the ulnar, radial and median nerve innervated hand and wrist muscles.

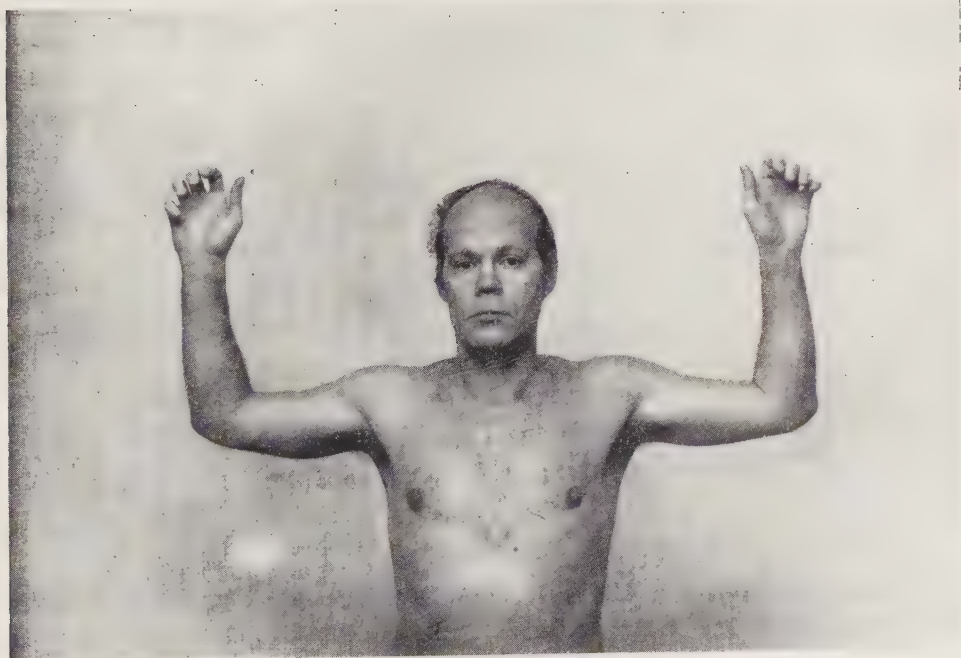


Fig. 1. The Abduction – External – Rotation (AER) position.

Biceps and triceps muscles strength was also tested. Hand grip strength was measured with a dynamometer (Collin adult) and graded in kp. The sensibility of the hand and forearm was tested with pin-prick, registering the sensibility in all dermatomes of both upper extremities. Supraclavicular tapping and thumb pressure for 30 s in the supraclavicular fossa over the brachial plexus was tested and the results noted. The elevated arm stress test (AER test, i.e. Abduction-External-Rotation-test, see Fig. 1) according to Roos (1976) was performed with the shoulders braced backwards and the patient clasping and opening his fists at a moderate speed for 3 min. During the test symptoms such as pain, early fatigue, tinglings or paraesthesias were registered as well as signs of venous congestion and changes of radial pulse. The costoclavicular compression manoeuvre, i.e. "military test" or Eden manoeuvre (Falconer & Li, 1962; Falconer & Weddell, 1943) was performed with the shoulders thrust downward and backward in an exaggerated military position and symptoms noted. This manoeuvre is known to close the costoclavicular scissor on the brachial plexus.

Tinel's test and Phalen wrist flexion test (Phalen, 1970) were performed in order to reveal absence or presence of median nerve compression (carpal tunnel syndrome). Percussion over the spinous processes in the thoracic and cervical spine and over the parascapular, paracervical and trapezius muscles were noted and graded. The head was also tilted to both sides and flexed and symptoms of cervical root compression noted. In all patients X-ray examinations of the cervical spine and thoracic aperture were performed and radiological findings such as cervical spondylosis, cervical ribs, previous fractures of the clavicle or first rib with callus formation, exostoses of the clavicle or first rib or the presence of anomalous first rib were noted.

Results

Three hundred and seventy-seven patients with brachial pain were examined, 237

Table 2. Correlation between symptoms and degree of brachial plexus compression and arterial compression as evaluated by Doppler flowmetry in patients with thoracic outlet syndrome (TOS) and patients with brachial pain, not related to TOS (non-TOS). (n)=number of cases. Figures in brackets represent patients with bilateral symptoms

Patients (n)	Symptoms of compression	Arterial compression, number of extremities				Tot. numb- er of extremities
		No	Slight	Moderate	Total	
7 (5) slight	} plexus	9	1	2	2	14
75 (21) moderate		67	11	26	46	150
155 (53) severe		111	23	44	132	310
3 (2) arterial	} plexus	1	0	0	5	6
8 (4) venous		7	0	0	9	16
248 (85) all TOS		195	35	72	194	496
129 (34) non-TOS		114	24	67	53	258

were judged to have compression of the brachial plexus and 11 isolated symptomatic vascular compression. The remaining 129 cases had symptoms not related to TOS. The differentiation of the total material into subgroups is presented in Table 2.

Out of 237 patients with nervous compression seven had only slight symptoms such as paraesthesias on prolonged elevation of the arm. Seventy-five patients had moderate symptoms with positional paraesthesias and intermittent aching pain and specific muscle weakness. One hundred and fifty-five patients had severe symptoms of brachial plexus compression with aching pain, muscle weakness, paraesthesias and dysfunction of the hand. Of the 11 patients with isolated vascular compression eight had pronounced venous compression with venous engorgement and cyanosis on elevation of the arm. Three of these initially presented with acute deep venous thrombosis of the axillary vein. Three patients had isolated pronounced arterial compression with pallor, coldness and arm claudication (see Table 2).

The most provoking position for detection of compression of arterial flow with Doppler technique was obtained with the arms elevated with flexed elbows, forearms either horizontal or straight up, and rotating the head away from the recording side, i.e. AER position (see Fig. 2). The other most provoking position

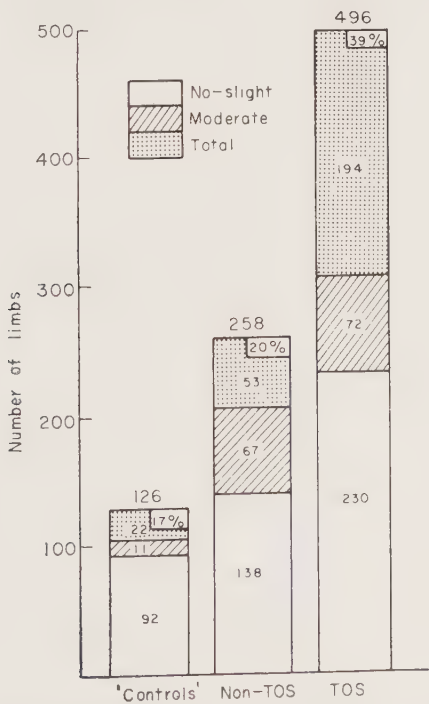


Fig 2. Arterial compression determined with Doppler-flowmetry in AER position in "controls", non-TOS and TOS patients. Numbers in columns refer to numbers of extremities and percentage total occlusion.

was with the arms straight up. The correlation between brachial plexus compression and arterial compression in the patients and "controls" is presented in Tables 2 and 3, respectively. A slight, but significant, correlation was established between quantitatively graded symptoms of nervous compression and the degree of the arterial obstruction as tested by the non-invasive Doppler flow test, $P<0.001$, with a specificity of 53 % and a sensitivity of 74 %. The presence of arterial compression assessed clinically and by Doppler flowmetry was compared. There was a significant correlation, $P<0.001$, with a sensitivity of 96 % and a specificity of 68 %.

Twelve patients had cervical ribs, three bilaterally. In these patients total arterial compression, assessed by Doppler technique, was present in 11 limbs (73 %) and in only four limbs (27 %) no compression was found. Concomitant compression of the brachial plexus was present in about 67 % of the patients with cervical ribs.

Table 3. Correlation between brachial plexus compression and arterial compression in 63 'control' individuals (126 extremities)

Plexus compression	Arterial compression, number of extremities				Total number of extremities
	No	Slight	Moderate	Total	
No	89	1	11	13	114
Slight	3	0	0	6	9
Moderate	0	0	0	3	3
Sum	92	1	11	22	126

Bilateral symptoms of severe and moderate plexus compression were present in 32 % (74/230) whereas bilateral involvement in the cases with symptomatic vascular compression was encountered in 54 % (6/11).

Symptoms from the upper extremities were almost twice as common in women as in men (Table 1). The mean age of the patients with symptoms not related to TOS (non-TOS) was somewhat higher, i.e. 41.1 years as compared to the mean age of the TOS patients, i.e. 37.5 years. This age difference was statistically significant at the 5 % level. No significant difference as to affection of the right or left arm was noted in TOS or non-TOS patients. The mean duration of symptoms was 2.7 years in the TOS-patients and in the non-TOS patients 4.1 years. In 129 patients with brachial pain other causes than TOS were present, i.e. cervical rhizopathy and myotendinitis of the shoulder girdle in 38 cases. Humeroscapular tendinitis and carpal tunnel compression were evident in 29 cases. Epicondylitis was found in one and entrapment of the ulnar nerve at the elbow in another two cases. In 54 patients we were unable to establish any specific cause of their symptoms but TOS could be excluded.

Discussion

Because of the close anatomical relationship between the subclavian artery and the brachial plexus as they leave the thorax together arching over the first rib between the anterior and the middle scalene muscle, there is an anatomical possibility of concomitant compression of both artery and plexus. A number of clinical tests have been introduced to evaluate the arterial compression. The simplest approach is to assess compression of the subclavian artery by evaluating disappearance of the radial pulse by palpation. Another possibility is the detection of bruits in the infraclavicular region during various provoking manoeuvres with different positions of the arm. In normal subjects positional compression of the subclavian artery is a common phenomenon (Dale & Lewis, 1975; Gergoudis & Barnes, 1980; Gilroy & Meyer, 1935; Raaf, 1955; Torriani, 1971; Wright, 1945). However, the prevalence of arterial and associated nervous compression has not been satisfactorily evaluated, therefore we have examined this relationship in a clinical material of cases with symptoms from the upper extremities and a limited control material. Our controls were selected from two small populations of young individuals without clinical symptoms related to vascular or nervous compression. The final analysis, however, showed not only the presence of arterial but also subclinical plexus compression in some cases. Due to the difference in age to the clinical material, this cohort cannot exactly be designated as a control group. Therefore we chose to refer to this group as "controls". In our study of 63 'control subjects' 17% (22/126 limbs) had total arterial compression. This is, however, less than reported by Gergoudis & Barnes (1980), who reported as much as 60% in a control population. The difference may be due to the method these authors applied.

Photoplethysmography essentially records pulsatile variations of blood volume in the subpapillary cutaneous plexus (Thulesius, Borgnis & Dvorak, 1973), whereas Doppler flowmetry in our study is a recording of arterial flow velocity. With the arms elevated there is bound to be a considerable degree of venous emptying and hence smaller signals will be the result. Other objective methods evaluating the arterial compression have been developed using oscillography (Dunant, 1976; Lo-A-Njoe, 1974) and phonoangiography (Dunant, 1976; Dunant *et al.*, 1974; Hehne *et al.*, 1975). Even with these tests positive oscillographic and phonoangiographic findings are frequent in normal subjects (Dunant, 1976; Dunant *et al.*, 1974).

The Adson manoeuvre (Adson, 1947), i.e. the patient takes a deep breath, elevates the chin and turns his head towards the affected side, has been widely used but it has been clearly demonstrated by Roos (1976), that a positive test has no relation to the presence of symptoms of TOS. The hyperabduction test, i.e. the extremities hyperabducted with the hands behind the head, as advocated by Wright (1945) provoked disappearance of the radial pulse in 83% of normal subjects. The shoulder-braced position of Eden (Falconer & Li, 1962; Falconer & Weddel, 1943), i.e. the costoclavicular manoeuvre, provokes arterial compression in more than 60% of normal subjects as shown by Raaf (1955).

Regarding invasive diagnostic tests it has been conclusively shown by Rainer & Sadler (1975) that positional arteriography has no bearing on the diagnosis of TOS and thus can not be used as a method of choice of patients for operation.

Our study has established a significant correlation between compression of subclavian artery and brachial plexus, but the Doppler test gave quite a number of false positive and negative results. The degree of arterial compression could also be assessed by palpation and auscultation, and every clinically significant compression of the subclavian artery could easily be detected by clinical examination.

It may seem surprising that the correlation between arterial and nervous compression is not more pronounced despite the close proximity of these structures. Compression in the thoracic outlet is, however, not only a simple mechanism, which can be explained by the "scissors" effect of the clavicle and the first rib. Much more important in the etiology of TOS seems to be the presence of congenital fibrous and fibromuscular bands mechanically narrowing the outlet, as discussed by Roos (1976). This applies to the most frequent type of clinically important TOS, i.e. compression of the brachial plexus, whereas vascular obstruction may be more easily explained by simple mechanical compression, such as the "scissors" effect. At the present time there is no proof of the assumption, but our results may suggest such an explanation, since nervous compression is more often unilateral and vascular compression more frequently seen on both sides.

Thus, assessment of arterial compression at the outlet, either by Doppler flowmetry, clinical examination or other methods, is not sufficient to establish the presence of compression of the brachial plexus. Clinically significant vascular compression was rare, i.e. 2.9%, but when present it was much more often venous than arterial, see Table 2. This is in accordance with other studies (Roos, 1976).

There was a difference in age on the 5% level between the TOS and the non-TOS patients, the TOS patients being younger. This was probably due to the fact that the symptoms of TOS were elicited earlier because of the underlying congenital anomalies, while diseases due to wear of different kinds after several years of working predominated in the non-TOS group.

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II

Cervicobrachial disorders in certain occupations, with special reference to compression in the thoracic outlet

by

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Abstract

One hundred and ninety-one workers in three different occupations were examined regarding presence of symptoms from the cervicobrachial region. 18 % of the workers (27 % of the female and 11 % of the male workers) had symptoms of compression in the thoracic outlet. However, only 2 % of all workers had pronounced symptoms of compression in the thoracic outlet.

In addition, 27 % of the workers (33 % of the female and 23 % of the male workers) had symptoms of other cervicobrachial disorders. Thus 45 % of the workers had symptoms from the cervicobrachial region.

The workers with symptoms of compression in the thoracic outlet were significantly younger than the workers with other symptoms of cervicobrachial disorders, $p < 0.01$. No significant correlation was noted between vascular compression and symptoms of compression in the thoracic outlet.

Introduction

It has been shown by several authors, that symptoms of cervicobrachial disorders correlate significantly to occupations with heavy workloads, repetitive straining or unnatural static positions of the arms (4, 5, 6, 9, 10, 11, 12, 13). Mechanization and automation of work tasks may result in occupational hazards causing an increase of symptoms of cervicobrachial disorders due to increased work rates and awkward work postures (6, 11, 12).

The thoracic outlet syndrome (TOS) is a symptom complex with pain, paraesthesias, numbness and early fatigue in the upper limb due to nerve compression. The prevalence of symptoms of TOS in a normal population is not exactly known. However, in a previous study (17) of 30 unselected young soldiers and 33 female nursing students, symptoms of compression of the brachial plexus in the thoracic outlet could be provoked in six, i.e. 9.5 %. Five of these were females and only one male. It is not known whether there is a correlation between symptoms of TOS and different kinds of occupation. In this paper, *Cervicobrachial disorders not due to TOS* (CD) is used as a collective term including symptoms in the neck, shoulder and upper extremities such as cervical spondylosis, myotendinitis of the shoulder girdle muscles, shoulder joint affections or carpal tunnel syndrome. The aim of the present study was to determine the prevalence of *cervicobrachial disorders* and especially *symptoms of compression in the thoracic outlet* (TOS).

Material and methods

Two hundred employees were asked to join the study but nine could not be examined. In most cases this was due to sick leaves which in no case was caused by cervicobrachial disorders. Ninety-two with heavy industrial work, 62 office workers and 37 cash register operators were interviewed and examined.

A. Heavy industry

Every third employee in a truck manufacturing company was randomly picked out and asked to join the study. The work tasks consisted of assembly-line work at increased rate, welding, drilling and turning. These tasks were often performed in awkward positions. Nine of the workers were women and 83 men. The workers had two 15 minutes rest periods, one before and one after lunch. The mean age of the workers was 34.6 years, with a range from 19–63.

B. Office work

All employees in a company dealing with telecommunications and data processing were asked to join the study. The work consisted of typing and editing technical texts, using word processing equipments. This work included using a keyboard and a display unit. Thirty-five of the workers were women and 27 men. The workers had two 15 minutes rest periods, one before and one after lunch. The mean age of the workers was 32.8 years with a range from 22–59 years.

C. Cash register operators

All cash register operators in three different supermarkets were asked to join the study. The cash registers were placed on the right side of the operators. Although the registers had been lowered the operators had to keep their right arms in half abduction and elevation demanding continuous contraction of the muscles of the shoulders, neck and back. The workers had a 5 minute rest every hour and 60 minutes after 4 hours. All the workers were women, with a mean age of 35.6 years, ranging from 20 to 63 years. Most women worked part time.

Clinical diagnosis

Interview. All employees were interviewed with a questionnaire about symptoms from the upper extremities, such as radiating pain, paraesthesias or numbness, as well as any previous cervicobrachial trauma. Time of employment and working hours were noted.

Physical examination. Each employee was examined to detect any signs of muscle weakness, early fatigue or soreness in the cervicobrachial area. The abduction external rotation test (AER) according to Roos (16) was performed by each employee. In the AER test the patient was instructed to abduct his arms 90° with his forearms flexed 90° and shoulders braced backwards. The patient clenched and opened his fists at a moderate speed for three minutes. During this test symptoms such as pain, early fatigue, tingling or paraesthesias were registered.

Tenderness over the brachial plexus in the supraclavicular fossa was also registered.

Vascular compression was assessed clinically by palpation of the radial pulse in the AER position and with the hands in the lap. At the same time auscultation infraclavicularly was performed to detect bruits in different positions of the arms.

Criteria of neurovascular compression in the thoracic outlet

Positional paraesthesias and numbness on prolonged arm elevation was classified as "mild" symptoms of compression of the brachial plexus in the thoracic outlet. Positional paraesthesias and numbness together with specific muscle weakness and pain indicated "moderate" compression of the brachial plexus, while constant paraesthesias, numbness and pain in combination with muscle weakness and dysfunction of the hand were symptoms of "pronounced" compression of the brachial plexus. Symptoms of *arterial compression* are ischemic pallor of the hand, coolness and pain in the arm.

Symptoms of *venous compression* are swelling, cyanosis and engorgement of the superficial veins across the shoulder.

Results

The workers were divided into three different groups, i.e. those with symptoms of TOS, those with symptoms in the cervicobrachial region not due to compression in the outlet (CD), and those without any symptoms in this region, see table I.

Table I Total material of workers differentiated into 3 groups according to presence of symptoms in the cervicobrachial region.

	No symptoms	Symptoms of TOS	Symptoms of CD
Female workers heavy industry n:9	2 (1 %)	4 (2.1 %)	3 (1.6)
Female workers office n:35	21 (11 %)	6 (3.1 %)	8 (4.2 %)
Female cash reg. operators n:37	9 (4.7 %)	12 (6.3 %)	16 (8.4 %)
Male workers heavy industry n:83	47 (24.6 %)	12 (6.3 %)	24 (12.6 %)
Male workers office n:27	26 (13.6 %)	0 (0.0 %)	1 (0.5 %)
Total n:191	105 (55 %)	34 (17.8 %)	52 (27.2 %)

Symptoms in the cervicobrachial region occurred in 60 % of the female workers (49/81) and 34 % of the male workers (37/110), making a total of 45 % of all the examined workers. Seventy-eight per cent (7/9) of the female workers in heavy industries, 76 % (28/37) of the cash register operators, 40 % (14/35) of the female offices workers and 43 % (36/38) of the male workers in heavy industries but only 4 % (1/27) of the male office workers had cervicobrachial symptoms.

Incidence of TOS symptoms

Thirty-four workers had symptoms of TOS on provocation, i.e. 18 % of all workers. Twenty-one had bilateral symptoms. However, only four workers, i.e. 2 % had pronounced symptoms.

Twenty-two of the 81 female workers (27 %) had symptoms of TOS compared to 12 out of 110 male workers (11 %). Comparing the different vocational groups, 17 % of the workers in heavy industry, 10 % of the office workers and 32 % of the cash register operators had symptoms of TOS.

Paraesthesias and numbness was present in all workers with symptoms of TOS, while pain in the arms and shoulders was present in 50 %. Twenty-six per cent of the workers had pain in the neck and 1 worker had signs of venous obstruction with congestion, cyanosis of the arm as well as dilated superficial veins across the shoulder at provocation.

Nine female workers and six male workers with symptoms of TOS had additional symptoms, such as myotendinitis in the shoulder girdle muscles and pain in the shoulder joint. In all cases these symptoms were slight to moderate.

Cervicobrachial disorders not due to TOS (CD)

Fifty-two workers had symptoms in the cervicobrachial region not due to compression in the thoracic outlet, i.e. 27 % of all workers. Bilateral symptoms occurred in 17 workers. Twenty-seven (33 %) of the workers were females and 25 (23 %) males. Details of the symptoms are presented in table II. Pain in shoulders and arms due to myotendinitis of the shoulder girdle muscles or diffuse myalgia and shoulder joint affections were the most common symptoms. The cervicobrachial disorders differed from TOS by a normal AER test and no pain over the brachial plexus at percussion in the supraclavicular fossa. Seven workers had previously received medical treatment due to cervical spondylosis, pain in the shoulder joint or pronounced Raynaud's phenomenon.

Table II Cervicobrachial disorders in 52 workers.

	Number of patients	Bilateral symptoms
Pain in shoulder and arms	26	11
Shoulder joint affection	14	3
Cervical spondylosis	7	
Carpal tunnel syndrome	2	
Raynaud's phenomenon	3	3

Signs of vascular compression

Infraclavicular systolic murmurs and disappearance of the radial pulse occurred in a number of workers in all three groups in provoked positions, i.e. the AER test, see table III.

Table III Number of workers with infraclavicular bruits or disappearance of radial pulse, percentage in parenthesis. Neutral position = hands in lap. n = number of workers.

	workers with no symptoms n: 105 (210 arms)	workers with TOS symptoms n: 34 (68 arms)	workers with other cervico- brachial symptoms n: 52 (104 arms)
Infraclavicular bruits			
Neutral pos	0	0	0
AER pos. (I)	50 (24 %)	17 (25 %)	21 (20 %)
Disappearance of radial pulse			
Neutral pos.	0	0	0
AER pos. (II)	29 (14 %)	11 (16 %)	11 (10 %)
Overlap between I and II	9 (8 %)	5 (7 %)	2 (2 %)

No significant correlation existed between arterial or venous compression and the presence of symptoms in the cervicobrachial region. Disappearance of the radial pulse in the AER position was present in 13 % (51/382 arms) of the total material but in no case was there any marked reduction of the pulse or infraclavicular bruits with the arms at rest. Venous compression of moderate degree was present in 4 % (8/191) of the total material.

Age difference and time of employment

The workers with symptoms of TOS were significantly younger than workers with symptoms of other cervicobrachial disorders, $p < 0.01$ (Student's t-test), see table IV.

The majority of workers with symptoms of TOS had been employed less than 7 years, compared to about 43 % of the workers with CD, see table V.

	Female workers heavy industry	Male workers heavy industry	Female workers office	Male workers office	Female cash reg operators
No symptoms n:105	26.8 (24–33)	35.6 (22–53)	32.2 (22–40)	34.9 (23–55)	33.1. (22–47)
Symptoms of TOS n:34	26 (23–29)	28.5 (19–44)	27 (23–34)	–	30 (20–41)
Symptoms of OCD n:52	32.3 (22–38)	41.5 (22–63)	45.5 (29–54)	59	41.1 (25–63)
Total material n:191	29 (22–38)	35.8 (19–63)	30.6 (22–54)	35.8 (23–59)	35.6 (20–63)

Table IV Mean ages of groups, ranges in parenthesis.

Table V Time of employment and cumulative number of workers

Time of employment	Symptoms of TOS		Symptoms of CD		Healthy workers	
	n	%	n	%	n	%
>4 years	12	35.3	14	26.4	43	41
4-7 years	20	58.8	23	43.4	64	61
7-10 years	32	94.1	42	79.2	89	84.8

Discussion

There are certain occupations with a higher incidence of symptoms of cervicobrachial disorders, i.e. cash register operators, typists, packers and assembly-line workers (3, 8, 9, 10, 11, 12, 13). In the present study 24 % of the office workers had symptoms of cervicobrachial disorders almost exclusively among the female employees. The prevalence of cervicobrachial disorders has been estimated to range from 4 % in management staff to 21 % in assembly-line workers (7, 8).

In the present study 47 % of the assembly-line workers complained of symptoms from the cervicobrachial region. The high prevalence in the present study may be explained by the heavy workload exerted on the workers and awkward work postures. Several authors have shown the negative effects of these two factors as well as monotonous working conditions and noise resulting in a higher frequency of cervicobrachial disorders (8, 9, 13, 14, 18).

Ohara et al (12) showed that 31.3 % of 371 cash register operators suffered from cervicobrachial disorders.

In the present study 76 % of the cash register operators had symptoms from the upper extremities. The operators had regular periods of rest and the electronic registers had been lowered so as not to strain the arms too much. In spite of these improvements the cash register operators had the highest incidence of symptoms of cervicobrachial disorders in our study. This was probably due to the continuous strain on the shoulders and arms and repetitive high speed motions.

Cervicobrachial symptoms, including symptoms of TOS, occurred twice as often among the female workers. Even among the female workers the incidence varied, i.e. almost 80 % of cash register operators and female industrial workers but only 40 % of the female office workers. Symptoms of TOS occurred in 27 % of the female workers, the female predominance being 2.5:1. These symptoms were present in every third cash register operator being twice as common as in workers in heavy industry and three times as common as in the office workers. This indicates that the prevalence is not correlated to the actual heaviness of the work but rather to the awkward work postures and amount of continuous muscle tension. Symptoms of TOS were most often bilateral in all vocational groups, whereas symptoms of other cervicobrachial disorders occurred to a greater degree on the dominant side among the male workers in heavy industry. This was probably due to excessive work load on the dominant side.

The female predominance of TOS is in agreement with other studies (1, 2, 15). Whether this is due to less developed muscles with more horizontal clavicles in females, a greater tendency for drooping shoulders or if congenital anomalies in the thoracic outlet are present to a higher degree is not known.

The incidence of TOS symptoms was 18 % in the examined workers, although pronounced symptoms were present in only 2 %.

As a comparison we found an incidence of TOS symptoms of 9.5 % in a control group of 63 young and healthy subjects.

There is, however, some doubt about these figures as an unknown number of workers continuously are transferred to less strenuous works or further education, due to symptoms in the cervicobrachial region. These workers are lost to examination as they have already been transferred before the study. This uncertainty is common to all studies of the same kind, however.

There was a significant age difference, $p < 0.01$, between workers with symptoms of TOS and other cervicobrachial disorders, i.e. the workers with TOS being younger. This is in agreement with other studies (2, 16).

The time of employment was shorter in the majority of workers with symptoms of TOS as opposed to workers with other cervicobrachial disorders. This seems to support the opinion that there is a fundamental difference between TOS and other cervicobrachial disorders, i.e. in almost all cases of TOS congenital anomalies are present making the person particularly susceptible to develop symptoms should the work contain provocative tasks or work postures.

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III

Physiotherapy in patients with thoracic outlet syndrome

by

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Abstract

Ninety-nine patients with symptoms of thoracic outlet syndrome (TOS) have been treated with physiotherapy. A common pattern of postural disorders were noted in all patients with TOS, and the patients were treated with correction of these disorders.

Twenty-six patients obtained a complete relief and 30 patients had a marked improvement but still some symptoms.

Five patients had only slight improvements and 38 patients obtained no relief of their symptoms. The severity of the symptoms was correlated to the results, i.e. patients with severe symptoms of TOS obtained relief in only 9 %, whereas patients with slight symptoms had relief in 83 %.

The result of the treatment was also correlated to the duration of the symptoms. Symptoms of long duration were less apt to be alleviated by treatment than symptoms of short duration.

Introduction

Compression of the neurovascular structure in the thoracic outlet region has long been recognized as a common cause of distressing symptoms in the upper extremities. Congenital anomalies such as cervical ribs and incomplete development of the first thoracic rib are well known (3, 6, 20).

Adson and Coffey were the first to point out the anterior scalene muscle as a possible cause of the thoracic outlet syndrome (1). This was further implicated by other authors (9, 11). Also acquired factors such as fractured clavicle or first rib, injury to the head, neck and shoulder region and hypertrophied muscles in athletes are well known. However, all these conditions are present in only a small number of patients with thoracic outlet syndrome.

In a cadaver study Roos found congenital fibrous or muscular bands in 33 % of the cadavers that might have affected the neurovascular structures in the outlet region (15). Furthermore, Roos found, in almost one thousand TOS-operations, that most patients had congenital bands predisposing them to irritation of the plexus (14). Nine different congenital bands have been recognized (16). Another factor of importance in the etiology of TOS is a poor posture.

The aim of this study was to see to what degree the symptoms of TOS could be alleviated by correction of the postural disorders, and the result of this treatment comprise the present report.

Material and methods

Clinical material

Of 271 patients originally referred to the surgical department with a tentative diagnosis of thoracic outlet syndrome during the period from 1979 to the first part of 1981, 99 patients were clinically found to have thoracic outlet syndrome. Sixty-seven patients were women and 22 men, the women/men ratio being 3:1. Bilateral symptoms were present in 45 patients. The mean age was 39.8 years ranging from 21 to 61 years. All the patients with thoracic outlet syndrome were referred to physiotherapy and reevaluated after termination of the treatment. The time lapse from the first examination to the reevaluation was 12.4 months ranging from 3 to 30 months.

All patients had a cervical spine X-ray. The X-ray was normal in 64 patients. Twelve patients had cervical ribs, 5 bilaterally. In only one case, however, the cervical rib was longer than 2.5 cm. Spondylitis and degenerative changes of the cervical spine was present in 33 patients, but the changes were slight in all but 2 patients.

Forty of the 99 patients had previously been treated with conventional physiotherapy such as hot packs, ultrasound, massage or by shoulder girdle exercises.

advocated by Britt (4). As only 5 of these patients had some benefit of this treatment it was decided to treat all these patients by correction of postural disorders.

Methods

Clinical examination

Patients with cervicobrachial symptoms were examined with a detailed history covering all symptoms of the head, neck and upper extremities followed by a thorough physical examination (18). The most reliable test of thoracic outlet syndrome was the three-minute abduction-external-rotation-test advocated by Roos (AER-test) which reproduces the patients usual symptoms (14). Another important test of compression of the brachial plexus was pain when tapping over the plexus in the supraclavicular fossa. After the diagnosis of TOS was established the patient was referred to physiotherapy.

Table I Number of patients with thoracic outlet syndrome, differentiated according to severity of symptoms. Figures in brackets represent patients with bilateral symptoms.

TOS	Patients (n)
Slight	36 (14)
Moderate	30 (21)
Severe	33 (10)
Total	99 (45)

Symptoms of thoracic outlet and clinical findings

The patients were differentiated according to the severity of their symptoms, see table I. Numbness and paraesthesias occurred in all patients, aching pain in 75 %. Weakness and fatigue in the arm were present in 38 % in patients with slight symptoms of thoracic outlet syndrome, the frequency increasing with the severity of symptoms to 100 % in patients with pronounced symptoms. Nocturnal symptoms such as pain and numbness were present in about 50 % of the patients with slight symptoms, increasing to 80 % in patients with severe symptoms of thoracic outlet syndrome. The mean duration of symptoms was 3.1 years (1 month to 12 year). Mild symptoms of TOS were paraesthesias and numbness after prolonged elevation of the arm. Patients with moderate TOS had positional paraesthesias and numbness but also intermittent pain in the cervicobrachial area and specific muscle weakness and fatigue. Patients with severe TOS were partially incapacitated from pain or paraesthesias, and dysfunction of the hand with muscle weakness, fatigue and clumsiness. These patients were usually more incapacitated from the pain than from the paraesthesias due to the pronounced plexopathy.

Previous trauma

Seventeen of the 99 patients had experienced trauma to the head, neck or shoulder region. Two patients had a clavicle fracture. Twelve patients had been involved in traffic accidents and suffered typical whiplash injury, while 3 patients had received blows on the shoulders and arms. Three patients had suffered repeated injury to the arm and shoulder region. None of these patients had had any symptoms of thoracic outlet syndrome before the accident.

Concomitant diseases

Myotendinitis of the shoulder girdle was present in 38 patients, in most instances of slight to moderate severity. Twenty-eight patients had slight symptoms of cervical rhizopathy while 2 had more pronounced symptoms. Less frequent were tendinitis of the humeroscapular joint and carpal tunnel syndrome, 11 and 9 cases respectively. Raynaud's phenomenon was present in four patients.

Physiotherapy

Anatomical changes as a plausible theory: In patients with poor postures locking of the sacro-iliac joints (SI-joints) with a backward tilt of the pelvis cause an active insufficiency of the gluteal muscles and a concentric contraction of the iliopsoas muscles which will eventually go into spasm. This contraction rotates the lumbar spine and the sacrum towards the unaffected side of the body. To keep the body balance the thoracic spine will rotate towards the affected side of the body. This causes an increased load on the costotransverse joint with radiating pain along the ribs due to the friction in the joints. By rotation of the thoracic spine the normal congruence between the scapulae and the rib cage no longer exists and the inferior tip and the medial border of the scapulae protract. This leads to a ventral rotation of the clavicle, i.e. the clavicles are displaced downwards thus narrowing the thoracic outlet. The backward tilt of the pelvis locks the normal body movements leading to a disturbance of the gait coordination with shortened step and minimal or nonexistent diagonal movements of the trunk. The normal movements of the shoulders and arms are limited of the same reason. Patients with a backward tilt of the pelvis often have pain in the thoracic spine as well as in the shoulder girdle muscles due to the compensatory rotation of the thoracic spine. Due to the tilted pelvis and the active insufficiency of the gluteal muscles these patients very often have shortening of the hamstrings, iliopsoas, rectus femoris and lumbar extensor muscles.

Postural disorders

All patients were examined regarding their posture and a common pattern of postural disorders could be noted. In the sitting and standing positions the pelvis was tilted backwards and the lumbar lordosis diminished or even substituted by a

slight kyphosis. The thoracic kyphosis was diminished and the upper part of the thoracic spine often "caved in" with a marked kyphosis in the transition to the cervical spine. The scapulae were protracted and hips and knees hyperextended in the standing position. In the walking position the pelvis remained tilted backwards and the diagonal trunk movements most often completely lost. Movements in the shoulder joints and arms were markedly diminished or absent and the patient walked with short steps. Because of the poor posture many patients had pain in the thoracic spine and shortening of various muscles, most often the hamstrings, iliopsoas or rectus femoris muscles.

Physiotherapeutic treatment principles

1. Reposition of the SI-joint and treatment of the iliopsoas pain.
2. Instruction for coordination exercises. This training is done by the patient preferably twice a day, for 30 minutes.
3. The coordination exercises are supplemented by other physical activities, such as long walks, skiing, skating, riding.
4. After a few weeks training, the muscular pains can be treated as well as back trouble for instance with ultrasound heat and soft tissue mobilization.
5. The patient is also given ergonomic instructions, which include posture training.

Results

The results of the physiotherapy are presented in table II. 26 (26 %) patients were completely recovered, although in 10 (10 %) patients their former symptoms could be provoked by the AER-test but at a lesser level than before the treatment. Thirty (30 %) patients were markedly improved but still had some mild symptoms. In 5 (5 %) patients there were only slight improvements and in 38 (38 %) patients the treatment could not relieve their symptoms.

Table II: Results of physiotherapy with correction of postural disorders according to severity of symptoms of TOS. The figures in brackets indicate number of patients with bilateral symptoms. n = number of patients.

Results	Number of patients with symptoms of TOS							
	slight		moderate		severe		total	
	n	%	n	%	n	%	n	%
Excellent	17 (8)	47	8 (5)	27	1 (1)	3	26 (14)	26
Good	13 (1)	36	15 (9)	50	2 (1)	6	30 (11)	30
Fair	1	3	2 (2)	7	2 (1)	6	5 (3)	5
Poor	5 (5)	14	5 (5)	16	28 (7)	85	38 (17)	38
Total	36 (14)		30 (21)		33 (10)		99 (45)	

The result of the correction of postural disorders in the 40 patients previously treated with conventional physiotherapy was not different from the result in the remaining group of patients. Thus 56 patients (56 %) obtained a marked improvement by the treatment. Differentiating the patients according to severity of their symptoms showed that patients with slight symptoms of TOS had the best benefit of the treatment.

The overall results of physiotherapy in patients with bilateral or unilateral symptoms were equal, but taken individually the outcome of the physiotherapy was more favourable if the symptoms were unilateral. The results of treatment were correlated to the duration of the symptoms. In patients who obtained a good result the mean duration of symptoms was 1 1/2 year, whereas patients with little or no benefit of the treatment had had symptoms with a mean duration of 4.3 years. The result of physiotherapy in patients with TOS after trauma is reported in table III. None of the 17 patients, with the exception of 2 patients, benefitted from the treatment.

Table III: Results of physiotherapy in patients with traumatic thoracic outlet syndrome, differentiated according to severity of symptoms. n = number of patients.

TOS	Trauma (n)	Results of physiotherapy excellent/good	poor
Slight	4	2	2
Moderate	3	0	3
Severe	10	0	10
Total	17	2	15

Twenty-seven of the 99 patients were later operated with transaxillary resection of the first rib. The decision to operate was made if the symptoms were severe and incapacitating in spite of 3 months physiotherapy.

Discussion

The existence of congenital anomalies in the thoracic outlet has been pointed out by Roos and in his report almost every patient operated for TOS had congenital anomalies (14, 15). Thus, the congenital bands seem to be important etiologic factors in TOS, but evidently the congenital anomalies can be present without evoking any symptoms, even for a lifetime, which has been shown by Roos in a cadaver study (14).

However, symptoms can be precipitated either by neck injury or spontaneously by increased muscle tonus due to poor posture. As the muscles go into spasm the congenital anomalous bands start tugging on the nerves causing pain and paraesthesias and further muscle spasm, and a vicious circle is established. By continu-

ing irritation on the plexus the symptoms deteriorate and will eventually pass the point where physiotherapy is likely to give any relief. The hot packs and gentle massage often make the patient feeling better but only for a very short time. Cervical traction almost invariably aggravates the symptoms of thoracic outlet syndrome and should be avoided (14).

By correction of postural disorders as described in the present paper many patients experienced an alleviation of their symptoms, but in a matter of 3–4 months the symptoms often were back although at a somewhat lower intensity. By resuming the treatment the patients could again alleviate their symptoms and keep them under control. There are several reports on good results of physiotherapy with a success rate from 50 to 90 % (2, 4, 5, 7, 8, 10, 12, 13, 17, 19, 21). In all these reports the patients have been treated with shoulder girdle strengthening exercises but only two authors have added correction of faulty posture. The severity of symptoms have been specified in only one report (2), where patients with moderate or minor symptoms were treated with physiotherapy.

In our study conventional physiotherapy or shoulder girdle exercises most often did not alleviate the symptoms.

The correction of postural disorders has shown to be effective in patients with slight or moderate symptoms with marked improvement of the symptoms in 83 and 77 % respectively.

However, in patients with severe symptoms of TOS or with a traumatic compression of the brachial plexus 85 % had no benefit by the treatment. In these patients the transaxillary resection of the first rib and congenital anomalous bands still is the method of choice.

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IV

Surcigal treatment of the thoracic outlet syndrome

by

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Summary

Sixty-three patients with severe symptoms of thoracic outlet syndrome have been operated with transaxillary rib resection. After a mean time of 2,5 years postoperatively the patients were re-evaluated. Sixty-four per cent had a complete relief of all their symptoms and 17 % had a marked improvement. In 12 % the results were classified as fair, while in 7 % no improvement of the symptoms was obtained.

Introduction

The thoracic outlet syndrome (TOS) is a compression of the neurovascular structures in the thoracic outlet area causing paraesthesias, numbness, pain and weakness in the upper extremities. Almost 98 % of TOS patients have symptoms of brachial plexus compression, whereas significant vascular compression is extremely rare, arterial compression even less frequent than venous compression, 0.5 % and 1.5 % respectively (Roos 1978). Because of the difficult diagnosis the syndrome has not been very well understood and thus the treatment has varied. In 1935 Ochsner et al and 1938 Naffziger and Grant considered the anterior scalene muscle to be the cause of the neurovascular compression (Ochsner, Gage & de Bakey 1935, Naffziger & Grant 1938). As the scalenotomy was a simple operation it soon became the method of choice. Increasing disappointment with the results of scalenotomy caused McCleery 1951 to propose removal of the costocoracoid membrane and subclavius muscle in addition to the scalenotomy (McCleery, Kesterson, Kirtley & Love 1951). Because of the mounting failure rate Clagett in 1962 redirected attention to the resection of the first rib (Clagett 1962). But it was not until Roos in 1966 described the transaxillary resection of the first rib that there was a marked increase of interest in the thoracic outlet syndrome, because the transaxillary approach was relatively easier and safer than other methods (Roos 1966). According to Roos the majority of patients with TOS have congenital fibrous or muscular bands that mechanically compress the brachial plexus and provoke the TOS symptoms. Nine different bands have been described by Roos (Roos 1976, Roos 1979). Excision of these congenital bands in combination with first rib resection is mandatory, to achieve a total decompression of the thoracic outlet.

Material and methods

Material

During 1975–1979 410 patients were examined for suspected thoracic outlet syndrome. The diagnosis of thoracic outlet syndrome was made by a very careful evaluation of the patient's history and symptoms followed by a thorough physical examination (Sällström & Thulesius, 1982). The severity of symptoms were graded as follows: positional paraesthesias and numbness on prolonged elevation of the arm was classified as "mild" symptoms, whereas additional specific muscle weakness and pain indicated "moderate" symptoms. Incapacitating paraesthesias, numbness and pain together with muscle weakness and dysfunction of the hand was classified as "severe" symptoms. Sixty-three patients were found to have severe symptoms of TOS, 30 patients with bilateral symptoms. Nine of these patients had severe symptoms, while 7 patients had moderate and 14 patients mild symptoms on the other side. Fifteen patients had leftsided and 17 rightsided symptoms. Thirty-six of the patients were women and 27 men, the women/men ratio being 1.3:1. Six patients had suffered an injury to the cervicobrachial region. Two patients had had a whiplash injury and 4 patients blows to the shoulder region. None of the patients had had any symptoms of TOS before the accident.

The mean age was 37.0 years, ranging from 13 to 61 years. All except four patients had paraesthesias and numbness in the arms and hands as well as weakness and early fatigue. Eighty per cent of the patients had nocturnal symptoms. One patient had a pronounced compression of the subclavian artery with pallor, coldness and arm claudication. The symptoms were provoked already at 15 degrees abduction of the arm from the body. In three patients, without previous symptoms of TOS, early operations were performed due to deep venous thrombosis of the upper extremity. The mean duration of symptoms was 2.8 years ranging from 1 day to 6 years. All except the three patients with deep venous thrombosis, were initially treated conservatively with physiotherapy. If the patients still had incapacitating symptoms after 3 months treatment they were scheduled for operation. Sixty-three patients were operated, 9 bilaterally making a total of 72 operations. All patients operated bilaterally had pronounced symptoms on both sides. In 70 cases transaxillary resection of the first rib was performed. Resection of cervical ribs by the supraclavicular approach was performed in two patients. All patients were re-evaluated at regular intervalls postoperatively (1, 3, 6 and 12 months). The mean time between the operation and the final re-evaluation was 2.5 years (15–69 months).

Preoperative vascular studies

Arterial compression assessed by physiological examination, i.e. with the Doppler technique, was present equally often in the affected and unaffected arm. Arteriography was carried out in 30 arms, where digital palpation and the Doppler studies

had shown a complete disappearance of the radial pulse with the arm in provoked position. The arteriogram was completely normal in 15 arms while complete compression of the subclavian artery could be demonstrated in the remaining 15 arms with the arm in provoked position.

Phlebography was carried out in 15 cases where the physiological assessment of the venous emptying had shown complete compression. This included the three patients with acute deep venous thrombosis. In provocative positions of the arm a marked reduction or absence of flow could be demonstrated in 9 cases, whereas in 6 cases the subclavian vein was completely normal.

Roentgenological findings

X-ray of the cervical spine was normal in 49 cases and in 10 cases there were slight to moderate signs of spondylosis and degenerative changes of the cervical spine. The remaining 3 patients had cervical ribs, one bilaterally.

Concomitant diseases

Nine patients had myotendinitis of the shoulder girdle muscles of mild to moderate degree. Carpal tunnel syndrome was present in 4 patients and humero-scapular joint pain in 5 patients. The symptoms were all on the affected side.

Indication for surgery

The decision to operate was made if the nonoperative treatment was unsuccessful and the patient had continuing symptoms. If the symptoms were severe, as in the three patients with venous thrombosis, early operation was indicated.

Method

Transaxillary resection of first thoracic rib

This method has been described by Roos (Roos 1966, Roos 1980, Roos 1981). The 90 degree thoracotomy position is important, enabling the surgeon to get a better view of the pertinent neurovascular structures. The wristlock position of the arm helps to visualize the nerves and vessels, but lifting should be done *only* when necessary or there will be an overdue stretching of the nerves with discomforting pain postoperatively. The incision should be placed at the level of the third rib to avoid getting into trouble with the axillary fat and lymph nodules. When developing the axillary tunnel to the first rib the intercostobrachial nerve will be found, most often in the second intercostal space, transversing the field to the arm. The nerve should be freed and held to one side, but if it becomes severely bruised during the operation it is better to cut the nerve to avoid burning and aching pain on the outer side of the arm.

In many patients the supreme thoracic artery and vein cross the first rib and should be visualized and clamped before the rib is freed to avoid troublesome bleeding. By finger dissection the anterior scalene muscle is freed and carefully divided with a right angled hemostat and scissors. The medial scalene muscle and the intercostal muscle are then pushed off the rib with elevators thus freeing the rib from vertebra to sternum and dropping the pleura to the second rib level. The tendon of the subclavian muscle is divided and the rib resected near the costal cartilage anteriorly and near the transverse process posteriorly keeping the T1-root out of the field with the root retractor. It is important to cut the posterior stump very short to avoid continuing compression on the nerve or the nerve might be caught in the callus formation around the stump if it is left too long. The stump is shortened with a rongeur. In order to do this adequately the nerve root is retracted gently preferably with a fiberoptic suction thus providing the surgeon with a bright light exactly at the desired location. When the rib is removed the surgeon has to make a careful search for congenital bands as it is mandatory to excise all bands to relieve the symptoms. The anterior scalene muscle is shortened 1–2 cm to prevent reattachment. As the phrenic nerve passes over the anterior scalene muscle medially it is important to be careful when shortening the muscle or the nerve can accidentally be cut. The pleura is then carefully inspected to detect any holes. Pneumothorax is due to a lesion in the parietal wall of the pleura and the only treatment needed is to place a drain into the hole in the pleura and suck all saline and air out after closing the wound. The drain is then clamped and a chest X-ray is taken. If the lung expands well and no air is left, the drain can be taken out immediately.

Assessment of the operative treatment

An "excellent" result is obtained if the patient gets a complete relief of all symp-

toms of TOS, whereas a "good" result indicates a marked improvement but the patient still has some symptoms although very mild. If the symptoms have been alleviated but still remain at a moderate level the result is classified as "fair". A poor result indicates that the patient did not obtain any relief of symptoms by the operation.

Results

The results of the operative treatment are presented in table I. Sixty-four per cent of the patients obtained complete relief while 17 % showed a marked improvement.

Table I Clinical assessment of operative treatment in 63 patients (9 bilateral making a total of 72 operations).

	Patients	%
Excellent	46	64
Good	12	17
Fair	9	12
Poor	5	7

In the last 30 cases we have looked for congenital bands in the outlet area. In 16 cases we found a type 3 band (see fig 1), in 5 cases a type 5 band (see fig 2) and in one case a combination of a type 3 band and a type 4 band (see fig 3). In the remaining 8 cases we were not able to identify any bands.

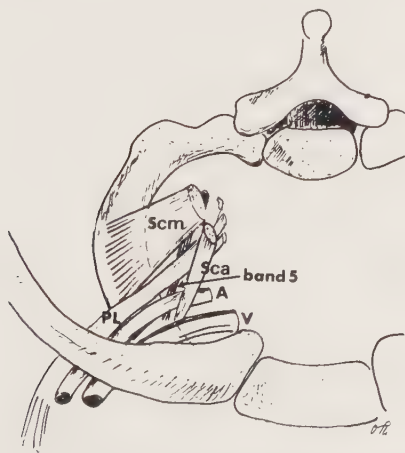


Fig 1: Type 3; a taut fibromuscular band from the neck of the first rib attaching to the rib behind the scalene tubercle.

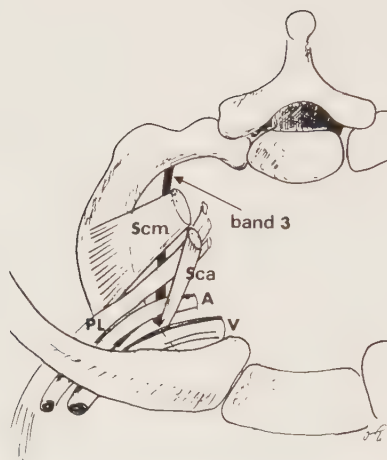


Fig 2. Type 5: the minimal scalene muscle attaching to the first rib behind the scalene tubercle.

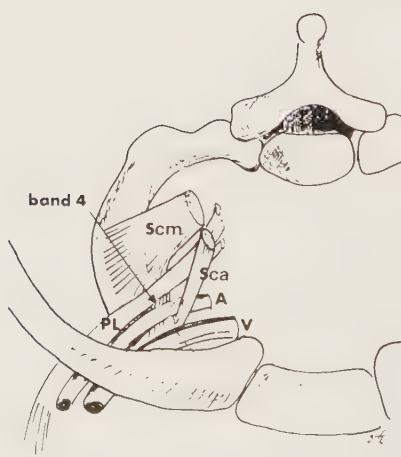


Fig 3. Type 4: a fibromuscular band from the middle scalene muscle to the anterior scalene muscle.

"Poor results"

Five patients had poor results. In 2 patients this was due to injury to the long thoracic nerve. One of these patients was operated due to acute venous thrombosis. The other patient, who was one of the first to be operated for TOS, was re-operated 3 months after the first operation because the posterior stump of the first rib was left too long. At re-operation the nerve was injured.

Three patients did not obtain any relief at all by the operations. Two of these patients had symptoms of cervical rhizopathy and one patient had shoulder joint pain, and all these patients were more than 45 years of age.

Complications

Complications are presented in table II. With the exception of the two patients with injury to the long thoracic nerve, the complications had no detrimental effect on the results of the operations.

Table II Complications to surgical treatment of TOS.

	Patients
Pneumothorax	7
Injury to the long thoracic nerve	2
Wound hematoma	1
Superficial wound infection	4

Recurrences

Three patients have been classified as recurrences after initially good results and two have later been subjected to reoperation by transaxillary neurolysis. The recurrences developed during the first 2 years after operation. One of the reoperated patients has obtained a marked improvement while the other patient still has pain and paraesthesias. The third patient is still on physiotherapeutic treatment with some improvement of her symptoms.

Concomitant diseases

The patients suffering from cervical rhizopathy had the same degree of symptoms postoperatively while 7 of 9 patients with myotendinitis showed a marked improvement of their symptoms.

The patients with carpal tunnel syndrome experienced no relief of their symptoms. This was true also for the patients with pain in the humeroscapular joints.

Discussion

The diagnosis of TOS is often very difficult. The process of diagnosis is one of elimination and is based upon history of pain, paraesthesias and motor dysfunction and a thorough clinical examination. Although a number of tests have been used to aid the diagnosis of TOS, such as electromyography or various vascular tests, the results have been disappointing.

The importance of vascular studies in patients with TOS has been stressed by several authors through the years, but it has become increasingly debated (Dale & Lewis 1975, Roos 1976, Roos 1978, Roos 1979, Sällström & Thulesius 1982). In the present study there was no significant vascular compression of the subclavian vessels in the affected arm except in four cases. In fact there was equally often no compression at all or very slight compression, both at the clinical examination and angiography or phlebography. Indeed, the benefit of invasive vascular tests in the diagnosis of TOS is very marginal. Invasive test should be used only when there are definite signs of marked compression even at rest, such as swelling of the arm or bruits infra- or supraclavicularly, prominent pulsations in the supraclavicular fossa, or more than a 15 mm of Mercury difference of the blood pressure in both arms. A cervical rib accompanied by vascular symptoms is also an indication for arteriography. Because nonoperative treatment is quite efficient in patients with mild or moderate symptoms and without risk, this should be recommended to these patients. However, if the patients are still incapacitated by their symptoms after 3 months of treatment, operative removal of the mechanical cause of irritation is the only treatment that can offer these patients relief. As the nonoperative therapy is unsuccessful in about 85 % of the patients with severe symptoms of TOS, it seems to be better to offer these patients operative decompression without initial physiotherapy (Sällström 1983).

There are various routes to first rib resection, i.e. the *transaxillary approach* (Dale 1975, Mc Gough 1979, Roos 1966, Roos 1980, Roos 1981, Urschel 1972), the *posterior parascapular thoracoplasty* (Martinez 1979), the *anterior supraclavicular approach* (Hempel 1980, Murphy 1980, Thomas 1979) and the *infraclavicular approach* (Nelson & Jensen 1970). Some surgeons use a modified transaxillary approach with the patient in a supine position with the arm lifted. For a total decompression of the TOS it is mandatory to resect the rib all the way to the transverse process, resection of the posterior one third of the rib being crucial as the lower trunk of the brachial plexus passes the rib in this region. Secondly excision of all congenital bands in the outlet area is mandatory to get a total decompression. The supraclavicular approach implies a very difficult and potentially dangerous dissection around the brachial plexus and the vessels covering the rib. In the infraclavicular route only the anterior two thirds of the rib can be removed safely. Moreover, the congenital bands cannot be dealt with by the infra- or supraclavicular routes.

Thus, these two approaches do not permit a total decompression of the TOS. With the dorsal parascapular approach the surgeon can make a total decompression, and this route differs from the transaxillary only by way of a higher postoperative morbidity due to the thoracoplasty.

Although the operative field is deep and tunnelshaped in the transaxillary approach it offers the best possibilities for complete resection of the first rib and all congenital bands. The neurovascular structures lie *behind* the rib, out of the operative field, as opposed to the supraclavicular approach. In the 90° thoracotomy position advocated by Roos (Roos 1966) the surgeon can look straight into the operative field whereas in the modified supine position the surgeon will have trouble getting the light into the field making the operation unnecessarily difficult.

We have personal experience with the supine position which was used in the first 15 cases. We had trouble getting sufficient light into the field making the operation difficult. Changing to the 90° thoracotomy position resulted in a considerable improvement of the visibility. In view of these facts we consider the transaxillary approach advocated by Roos to be the best route for first rib resection.

When the congenital bands are taut they cause a persistent irritation of the brachial plexus. The irritation causes an inappropriate stimulation of the nerves, eliciting muscle spasm and pain, and a vicious circle of muscle spasm, pain and irritation of the nerves is established. This is one of the main reasons why a majority of the TOS patients with concomitant myotendinitis get relief from their muscular symptoms following resection of the first rib and anomalous bands.

Cervical ribs occur in about 0.5 % of the normal population and in 10 % the cervical ribs are symptomatic (Crimm 1952, White, Poppel & Adams 1945). In the present study cervical ribs occurred in 6.5 %. According to Roos (Roos 1971) operation for a cervical rib in patients with TOS should always involve resection of the first thoracic rib simultaneously. Two patients with cervical ribs were operated with resection of the rib alone leaving the first thoracic rib in place. This is probably the reason why these patients did not obtain complete relief.

The results of operation for TOS vary from 57 % to over 90 % of the patients having good or excellent relief of their symptoms (Dale & Lewis 1975, Dunant 1976, Lo-A-Njoe 1974, McGough, Pearce & Byrne 1979, Roos 1979). Fair or poor results were obtained in 3–20 % of the patients. The differences in the results can be due to several reasons, one of which is technical, i.e. leaving either too long a posterior stump of the first rib or congenital bands that keep on tugging at the plexus. Another reason is that the diagnosis of TOS is sometimes very difficult and this is probably the answer to most of the poor results, i.e. incorrect preoperative diagnosis. Increasing personal experience by examining many patients with TOS is the best way to reduce the number of poor results.

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